BRAIN.

JANUARY, 1880.

Original Articles.

CHRONIC MORPHINISM.

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WHILE the progress of science is ever discovering new methods of combating disease, the advance of civilisation brings with it new enemies. Railways bring in their train spinal concussion, telegraphs develop telegraphic-cramp, and many valuable remedies become by their abuse the sources of new ailments. Such are the various forms of chronic poisoning, the prototype of which is chronic alcoholism, faithfully portrayed by Seneca. Specially worthy of notice in this relation is the continued abuse of chloroform, and opium and its alkaloids, more particularly morphia. It is different with nicotin, which is taken without the slightest evil effect by a large percentage of mankind; or even with arsenic. Chloroform and morphia differ from alcohol, inasmuch as the latter may be taken in considerable quantity without appreciable injury, so far as the nervous system is concerned; whereas the former two very frequently produce evil effects, and lead to earnest desire on the part of the individual to shake off the habit, which is at all times difficult and often impossible.

I will here confine my remarks to chronic morphinism, which is more common than chronic chloroform-poisoning. It

has to us a particular interest, inasmuch as quite an incredible number of our colleagues have fallen victims to it; and many have only just escaped. If medical men are charged—and it is to be feared, justly—with the propagation of this disease, owing to their carelessly, or for mere convenience' sake, leaving morphia and a subcutaneous syringe with the patient, it may be regarded as their punishment that the demon morphinism finds among them his favourite victims.

The number of cases hitherto published is not so great as to render unnecessary the description of others, yet they are sufficiently numerous to afford a tolerably accurate picture of the disease, and to indicate its treatment and prognosis.

As it is not my intention to write an exhaustive monograph on morphinism, I will content myself with calling attention to a few points, on the basis of my own and other observations, and in particular to those which at present are less generally known. The first commencement of the use of morphia is almost always some painful ailment; and then, as the pain continues, or even though it may have disappeared, the habit is formed, and more and more of the drug is demanded.

So began one of my patients, after traumatic injury of the sciatic; another in consequence of periostitis; a third (a physician) for toothache; a lady took to subcutaneous injections to allay violent pains in the sacrum caused by pelvic disease. In other cases, however, purely nervous symptoms lead to the formation of the habit. As is well known, we have in the subcutaneous injection of morphia a powerful weapon against feelings of fearful anxiety; so that states of melancholia, even of high degree, not unfrequently give way before it. Such intense anxiety and insomnia led in two of my patients to the use of morphia. But other nervous conditions lead to a similar result. Thus a lady exhausted by long-continued vomiting of nervous origin, found opium the only remedy of any avail. Though the existence of some painful affection is the chief cause of morphinism, yet there are others. A young medical man gave the following account of his own case. While he was attending the hospital, a patient was dismissed suffering from carcinoma of the stomach, and who had been for a long time treated with subcutaneous

injections of morphia. Next day the patient returned in a state of great excitement, and piteously begged for an injection, as otherwise he must die. This occurred in 1869, at a time when chronic morphinism and its phenomena were less known than now. As the physician was inclined to believe that the patient was romancing, he tried the experiment on himself to ascertain what the effects were. The result was that he formed the habit of morphinism, and never could overcome it.

With reference to the *quantity* taken, it is to be noted that it varies within certain limits, and may not exceed the maximum. The smallest quantities which can be taken continuously without any very marked effects, or reaction, are variable with the individual. I have under treatment a lady who has taken daily for more than a year about 0·003 gramme, and who has never felt inclined to increase the dose, though she strongly objects to giving it up entirely, on account of restlessness, sleeplessness, &c. While in most cases of continued morphinism a habituation occurs, so that more and more may be taken, there is a limit, variable in different individuals, beyond which, as a rule, it is impossible to go. If the morphinist has reached the maximum dose, he is aware of it, and instinctively keeps on this side of it; so that we very seldom hear of acute poisoning with morphia in such cases. There are individuals who never exceed 0·005 gramme daily, and have similar symptoms to those who consume 2–2·5 grammes. More than 3·5 grammes may not be taken daily continuously. This appears to be the limit, even in the most pronounced cases.

With respect to the symptoms of morphinism, they are so well known that they do not require to be detailed at length. If a morphinist who comes for advice and help is asked what symptoms he chiefly complains of, the answer is much the same in all cases. Disinclination to exertion in general; apathy; loss of memory; restlessness; generally also loss of appetite, various pains, hyperæsthesia; and in pronounced cases, suicidal tendencies. The patients are feeble, emaciated, have an ashy-grey or livid complexion, yawn and sneeze a great deal, and are impaired as to sexual power or completely

impotent. Less attention has been paid to the psychical condition. In this there is a certain analogy between alcoholism and morphinism. As the former is characterised by a moral deterioration, or moral starvation—whence the name formerly given, "*inhumanitas ebriosa*"—so in the morphinist there is a moral decadence in many cases. As he endeavours to conceal his habit in every way, so he loses his respect for truth and tells lies to conceal his condition, and afterwards from mere habit. He even lies to his physician; one day he says he has been accustomed to take 0·5 gramme daily, and next day he makes it 1 gramme. From this tendency to lying, which, however, is not all to be put to the account of morphia, he soon loses all self-respect; he feels how deep he has sunk, and, seeing no means of escape, he sinks deeper and deeper. This is, of course, not true of all cases; but knowledge of this fact is necessary in order rightly to estimate the utterances of morphinists.

The phenomena of the *morphia-hunger* vary with the individual and with the duration of the fast. Very often, where the quantity is slowly diminished, there are, with the exception of increasing discomfort, no violent symptoms. When, however, it is entirely withdrawn, or nearly so, violent symptoms occur. These are acute diarrhœa; insomnia; great excitement, or even mania, dangerous to those around, and particularly the physician; pains, itching of the skin, perspirations, a feeling of coldness, hallucinations, collapse, &c. In general, the gradual withdrawal has little advantage over the sudden. When great care has to be exercised on account of complications (great weakness, great excitability, &c.), it may be had recourse to rather than the other; yet, as has already been remarked, the explosive symptoms may occur nevertheless.

To relieve the sufferings of the worst period in the weaning process, various methods have been recommended; but unfortunately, though often useful, they are not so in all cases, e. g. wine in tolerably large amount, bromide of potassium, warm baths. In some cases, packing in lukewarm wet sheets was of great service. It may be remarked also that, when the drug is gradually withdrawn, it is often of advantage to inject the

subcutaneous same quantity of liquid as before. Occasionally even the injection of pure water has been of benefit.

The *prognosis* of morphinism as regards cure is not so favourable as was at one time believed.

Several considerations influence the result.

(1) The duration of the habit. Those cases which have lasted only a few months are more easily treated, and with greater certainty against relapse, than those which have gone on for several years, subject to the deleterious influence both on body and mind.

This requires no further explanation.

(2) When the condition which originated the use of morphia continues, or is incurable, a cure of morphinism can scarcely be looked for.

(3) The physical, but more especially the nervous, constitution plays an important part. Very weakly individuals fall into such a state of prostration on withdrawal of their accustomed drug, that it is necessary to give it again. In other cases where there is a strong predisposition to nervous disorder, the withdrawal of the drug gives rise to intense psychical disturbances.

(4) The maximum dose taken daily is not of great importance in relation to prognosis. The tolerance of different individuals varies so much, that what is a large dose for one is only a moderate dose for another.

Attention to these points may indicate what are the prospects of cure in a given case, but by no means with certainty. On the contrary, many morphinists go away apparently cured, and full of gratitude to the physician who has freed them from their misery; yet, as the subsequent history shows, the cure has been merely apparent. On this more hereafter.

Cases are sometimes met with in which the symptoms consequent on withdrawal of the drug are so serious as to necessitate its re-administration. As a rule, however, these symptoms should not be allowed to become too intense, and an allowance of a smaller quantity of the necessary drug enables us to dismiss such a patient, at least better, though not cured. In the great majority of cases, however, it is possible to withdraw the drug entirely; but then the question

arises—with what benefit? In those cases which, according to the points mentioned above, allow of a favourable prognosis, it is frequently possible to effect a permanent cure; but if all the conditions are not fulfilled, the prospect in my experience is distinctly gloomy. Very often a relapse occurs after a short interval, and the individual returns to his old ways. In the worst cases, mental disorders, frequently characterised by diverse hallucinations, but more especially of a melancholic form, with pronounced suicidal tendencies, are apt to ensue. In reference to relapses, the frequency of which Erlenmeyer ('Centralblatt für Nervenheilkunde,' No. 22, 1879) has recently noticed, I may mention the case of a medical man who, after four attempts at weaning, was treated in our institution, and dismissed apparently well, and who yet after three months was again in his former state. Many of the patients from whom the drug has been entirely kept for a length of time fall into a state of apathy, mental incapacity, and purposelessness, still retaining those moral characteristics already described. Among the eight cases described below there are three in which the results were suicide by gunshot in two, and in the third—a lady—repeated and most determined attempts to make away with herself. Hence the physician who undertakes to wean a morphinist of his habit must be very careful both as to his prognosis, and as to his means of carrying out the treatment to its end. Not unfrequently the patient will be most benefited by a diminution of his quantity, while a complete withdrawal will produce only other disorders. Very strict supervision of the patient for some months after active treatment is also highly desirable, though not always easily carried out. Indeed, in many cases in which there is physical or other discomfort rendering life otherwise unendurable, it is better to continue the administration of morphia, than interfere with the habit. The following example will illustrate this.

A medical man, aged 52, a Pole, was at the end of 1878 in a great state of alarm and fear of infection by the plague, so that he could no longer attend to his work, and became depressed and lachrymose. In spite of various remedies, such as bromide of potassium, chloral, &c., his symptoms increased;

he had intense precordial anxiety and sleeplessness, tore his hair, and felt inclined to commit suicide. On admission into our institution on May 20, 1879, he was in a state of great excitement, weeping, howling, and, while detailing his woes, would suddenly start as if terrified at something

This condition continued the whole day, till 8 in the evening. At this time he became quiet and spoke rationally, exhibited some appetite, and felt quite well. Next day, however, the old condition returned. After various means had been employed, morphia injections were had recourse to. Twice or thrice a day, as seemed necessary, 0·01–0·02 gramme was injected subcutaneously. The effect was very marked. He became quieter and more hopeful, and gradually the quantity of morphia could be diminished. On July 31 he left the institution, but remained under supervision in the neighbourhood for three months longer. By the continued use of about 0·02 gr. morphia daily, he was more cheerful and contented, his weight increased, and his general appearance improved. His love for, and ability to work, however, have not yet come back.

Morphinism may be of medico-legal interest. The moral degradation which accompanies morphinism, like alcoholism, may lead to actions which bring the individual into collision with the law. It is not likely, however, that such a condition would be considered as a ground of irresponsibility. On the other hand, a case of simulation of morphinism lately occurred in Vienna. A young man was apprehended in the act of picking the pockets of two ladies at the same instant in a crowd. He excused himself on the ground of being a morphinist, and that through this "his cerebral blood-vessels were so congested that his ideas of things were perverted." He offered to take 1·0 gramme morphia, at one dose, in order to show, by his bearing it, that he was a morphinist. In the course of further observation by the police surgeon it became clear, however, that the accused could not simulate the phenomena of morphia-hunger; and that the plea was entirely false, and put forward merely to screen a case of wilful theft.

A series of experiments were made on animals in order to study the phenomena of morphinism in them, but though I

have carried on experiments for four months, I have not obtained any very characteristic results. I injected daily in a number of rabbits a quantity of hydrochlorate of morphia, beginning with 0·015–0·02 gramme, gradually increasing up to 0·3–0·4 gramme.

First it was observable that a similar habituation occurred in rabbits as in man. The animals treated by a gradually increased dose bore large doses of the drug much better than others. The symptoms observable after long-continued injection were, a certain want of tone and weakness of the muscles, most marked on the hinder extremities; and also trophic affections of the skin. In many of the animals the hair began to fall out, particularly on the neck, so that the skin became quite denuded in patches.

I could not make out any permanent contraction of the pupils; the vessels of the ears were shortly after the injection contracted, but later became dilated. In several animals, in order to ascertain if anything like morphia-hunger occurred, after three months daily injection—amounting at last to as much as 0·3–0·4 gramme—the administration was suddenly stopped. I cannot, however, say that I noticed any symptoms worthy of note. Perhaps one or more exhibited a slightly-increased reflex excitability, but within very narrow limits.

Owing to the comparative unimportance of the results obtained, I have not thought it necessary to record the experiments in detail. I will only mention that many rabbits, at varying periods in the course of the experimentation, i.e. in the first few days, or after several weeks, &c., died somewhat suddenly in convulsions. On section, the brain was found œdematous and swollen, while the other organs presented no discoverable abnormal appearances. Where such were visible, they were certainly not attributable to the morphia.

In conclusion, I append a few cases of chronic morphinism, which have been treated in my institution, with the remark that the first three cases have been already alluded to by Leidesdorf in his valuable work.

Case 1. C——, aged 35, formerly lusty and cheerful, had, a year before his admission, taken to morphia in consequence of acute periostitis. He rapidly increased the dose, and on

many days he had taken 80 injections of 0·02 gramme each. As the effect of the injections, after which he felt fresh and strong, wore off, and the more rapidly each time while the reaction became more intense, he resolved to place himself under treatment, in order to wean himself entirely. With this view he was treated by an able physician, and with success. But immediately after the weaning, he fell into a condition of melancholia, which rapidly increased. He fancied himself a wretch, who had ruined the whole world; felt in a constant whirl, and could give no account of what passed before him. His memory was very defective; he was restless, particularly at night, when black thoughts drove him from his bed. On the journey he made a suicidal attempt, inflicting a wound two inches long on his throat with a razor, with the purpose, according to his own statement, of relieving the congestion of his head. He was brought to our institution. His symptoms improved so rapidly, that after six days his wife insisted, contrary to medical advice, on removing him. We learnt that a few months afterwards he had shot himself in the Tyrol.

Case 2. S——, aged 33, had begun morphia injections seven years before admission, in consequence of injury of the sciatic nerve. Though the pain had speedily subsided, he had continued the morphia, and had gone on increasing the dose up to 2·0 gramme daily. Numerous abscesses resulting from the injections compelled him latterly to take the morphia chiefly by the mouth. He imported this direct from England in large original bottles, containing 100 grammes, and he took it with a spoon without dissolving it. The quantity which he took in this way must have amounted to nearly 3 grammes daily. Several attempts to wean himself having failed, he placed himself under our care. He had a pale-greyish complexion, and flabby muscles; but he was otherwise well nourished. He complained of apathy, and bad memory. He received on the first day 2·0 grammes, then 1·5, then 1·0, and on the fourth day 0·5 gramme morphia; while on the sixth it was entirely suspended. At the same time warm baths, strong wine, and at night beer was prescribed. Already at the diminution of the dose he was restless, complained of alternations of heat and cold, diarrhœa, prostration; and he could

obtain no sleep, even with chloral hydrate. Four days after the discontinuance of the morphia, a condition of collapse came on, so that it became necessary to give him an injection of 1·0 gramme. After this, and under the influence of bromide of potassium, the symptoms slowly improved, the appetite returned, but sleep for a long time was very poor. After eight weeks he was discharged; but according to later accounts he continued unable to turn himself to any employment, though he kept from morphia.

Case 3. R——, aged 30, is the case already mentioned (p. 451) of the physician who became a morphinist from experimenting on himself with regard to morphia-hunger. At first he took very moderate quantities, but later, becoming acquainted with another medical man also a morphinist, he increased his allowance. This other medical man also came under my treatment. (Case 4.)

The reason of R.'s coming to the institution was neither the inconvenience of the abscesses caused by the repeated injections in the thighs, nor the costliness of the drug, but, as he truly said, owing to a moral "Katzenjammer:" a feeling that he was not as other men, but had artificially to be kept up to the level. Before he came under my treatment he had four times tried to wean himself, but each time unsuccessfully. First, voluntarily, while he was still attached to the hospital; second, involuntarily, when he was unable to return from a journey and had to remain two days without a supply; third, at a hydropathic establishment in the Steiermark; and lastly, at a vegetarian institution in Switzerland. Latterly he had been in the habit of injecting about 2·0 grammes daily. On one or two occasions he happened to puncture a vein with his syringe, so that the morphia was injected directly into the blood. A strange itching sensation immediately overspread the whole body, and great congestion and a feeling of bursting of the head came on, with red, cyanotic face, giddiness, and laboured breathing. In the course of from 2–10 minutes after the injection the symptoms generally subsided. The quantity was diminished gradually each day, so that on the 8th he received none. The phenomena of morphia-hunger then showed themselves in all their intensity. He was highly

excited, threatened to make away with himself, declared that he was illegally detained, and swore he would call the police to his assistance. He broke the furniture; ate almost nothing for some days; complained much of cold, especially towards evening when he went to bed; violent diarrhœa; the pulse, which was formerly usually 72, sinking to 54. Then occurred hyperæsthesia and paræsthesia of the various senses; everything smelt and tasted bad. He was ordered bromide of potassium, wine, and warm baths. Gradually he became more quiet; his strength improved, but he continued for a long time very irritable and pugnacious. His pulse rose to 100. He began again to smoke and to seek amusement. He went away contented and happy and full of gratitude, after four weeks' stay in the institution. The first accounts were favourable enough, but half a year later he had already gone back to his old ways, and was as bad as ever.

Case 4. H——, aged 32, a medical man, had taken morphia for five years, having commenced it in consequence of toothache. As he found the morphia relieve also some mental troubles and worries, he continued with it. He gave also as another reason that, being assistant at the Physiological laboratory, he saw in himself an interesting subject of experiment. He found the internal administration better than the subcutaneous, though the action was less rapid. Various attempts to give up the habit failed. He took daily 1·2–1·3 grammes, and had also induced Case 3, whom he recognised as a morphinist by his aspect, to indulge more deeply than he had been wont.

The symptoms which he chiefly complained of were copious perspirations and pharyngeal catarrh. The ashy-grey complexion, peculiar to morphinists, was in him exceedingly well marked.

For the first few days of his stay in the institution he received from 0·6–0·2 gramme daily. On the sixth day it was entirely suspended. Then the phenomena of abstinence exhibited themselves in an intense degree. He was very excited, and wanted to climb up the wall with his feet. He had violent sneezing, sweating, and diarrhœa. Sacral pain; a feeling of coldness, chiefly one-sided; yawning, formication in

every part of the body, were the chief symptoms, in addition to great prostration. It was noticed that he very soon exhibited a profound aversion to morphia.

The treatment was similar to that of the former cases. Cherry-laurel water was very useful in calming his restlessness. His sleep was at first very indifferent, but improved under morphia, though he affirmed that he did not sleep at all, which was not really the case. His complexion became again fresh and ruddy. His desire for life and enjoyment returned, and, contrary to our advice, he went to the theatre on the 18th day, and left the institution at the end of three weeks apparently quite cured. He went to Italy, and returned after two months cheerful and happy at the successful cure which had been effected. He stated that during his sojourn in Italy, being attacked with diarrhœa, he had taken thirty drops of opium, a dose which he had found sufficient during his morphinism. This, however, was said to have produced symptoms of poisoning. He took up his residence in Vienna, but shot himself fourteen days afterwards. Shortly before, as we learnt on inquiry, he had on several occasions taken large doses of morphia.

Case 5. R——, aged 47, farmer. The patient's father was insane. The patient himself was formerly subject to headaches. Nine months before his admission into our institution, in consequence of overwork and worry, he began to suffer with gastric catarrh, palpitation and despondency. He could no longer maintain his family, could not work, was sleepless, and had creeping sensations all over his body, as if all the ants in the world had been let loose on him. All remedies proved of no avail; chloral was useless; morphia never relieved him bodily and mentally. He injected daily 0·15–0·02 gramme. Under hydropathic treatment in Gräfenberg he improved still further, but he was advised also to give up morphia entirely before he could be considered cured. Under the existing conditions, however, a weaning process seemed neither necessary nor likely to succeed, but at the very earnest desire of R. himself he was admitted as a patient. During the last few days he had taken what for him was a considerable dose daily, viz. 0·35 gramme, and had the symptoms of acute

morphia-poisoning on his admission—constipation, giddiness, somnolence, hallucinations of hearing. For the first twenty days of his stay he received morphia injections of gradually diminished amount, yet he very soon began to exhibit symptoms of morphia-hunger—formication, cold, diarrhœa, restlessness; and all the symptoms for which he took morphia came back. He complained of terrible dreams, and when in bed kept his legs in constant motion. The symptoms showed themselves with special intensity when the morphia was completely suspended. The psychical symptoms chiefly predominated. He threatened to prosecute everybody, beat his attendant, looked very wild, swore he must die, could not endure life longer. A violent pain extended from his hypogastrium through his limbs, and he complained also of pain in his joints and on both sides of his head. By means of chloral and bromide of potassium, administered chiefly as enemata, sleep could usually be induced. During the ten days of his abstinence the symptoms rather increased than diminished; he refused to obey instructions, and became ultimately so weak and unmanageable that morphia had to be again allowed. At first he received 0·15 gramme twice a-day; but as this did not prove sufficient, it was increased to 0·09 gramme daily, given in three doses. Hereupon the psychical symptoms disappeared, with the exception of slight hypochondriasis; and his bodily condition improved, particularly his gastric catarrh. He left the institution, but returned after his daily dose had reached 0·18 gramme, which was in the course of three weeks. He did not, however, enter the institution, but lived in private under the care of my assistant, Dr. Krueg. Under his treatment the quantity was reduced to 0·09 gramme daily, and with this the patient continued free from his troublesome symptoms, and returned home.

Case 6. M——, aged 33, a medical practitioner, had begun morphia seven years before, in consequence of periostitis of the lower jaw. He had, however, ceased taking it, but recommenced on being attacked by pleurisy, and had gone on, till lately he was in the habit of injecting as much as 0·6 gramme daily. He noticed, however, that he became less fit for work, that his energy failed, and therefore he resolved to

wean himself. On his admission the morphia was entirely suspended at once. For the first two days of abstinence he felt quite well, but sneezed a great deal. In the night of the third day he was seized with violent vomiting, repeated about 80 times within the 24 hours. The quantity of vomited matters was enormous, so that in this one day he became very low and prostrate, felt cold, and complained of sacral pains. The pulse, which at first was 76, sank to 46. Next day the vomiting ceased, the pulse was 42; there was great prostration, with diarrhœa. The colour of the stools was, as it is generally, very dark, almost black. From this time he recovered rapidly, so that already on the eleventh day he left the institution. His pulse was then 90. As to his subsequent history I have heard nothing.

Case 7. Frau S—— had taken morphia for two years, in consequence of a painful abdominal affection, and had come to taking 0·2 gramme daily. Under the morphinism her general health was deteriorating so much that she resolved to place herself under treatment for the purpose of being weaned. For the first two days a little morphia was allowed, but she already at the diminution of the dose complained of pain in the sacrum, general malaise, tremor of the limbs, creeping sensations all over, cold, dislike to food. She was very restless, yawned a great deal and could not sleep. These symptoms became more intense on the third day, when the drug was entirely suspended, but after two days, improvement occurred. Lactate of soda (10 grammes) once procured a few hours' sleep, but on the following nights proved of no use. For a few days she continued very irritable and excitable, but was dismissed as cured in a fortnight. She made a tour to Italy, and returned at the end of six months, quite fresh and blooming.

Case 8. Frau S——, a highly intellectual lady of nervous constitution, had been troubled for several years with nervous vomiting, which occurred particularly in the morning; and also with intense headaches. Of all remedies, morphia gave the greatest relief. Taken internally, it had scarcely any effect, but was active when injected subcutaneously. She stated that during the injection she often felt the blood

rush to the head, and that she often experienced a peculiar metallic taste in the mouth, which increased during the first 40–50 seconds, and then gradually faded away. Gradually she raised her allowance to about 2·0 grammes daily. She had made repeated attempts to wean herself, but unsuccessfully, as the above-mentioned nervous symptoms returned. She affirmed she even had an aversion to morphia, but could not do without it for the reasons mentioned. When she had reduced herself to 0·02 gramme daily, she suffered so much that she was obliged, contrary to her desire, to increase the dose again.

From this habit she had acquired a sodden sallow complexion, and a peculiar look of illness about the eyes. It is to be noted that she herself said that no one should trust a morphinist, as they were all alike untruthful. Such was the condition of things six months before her admission into the institution. Shortly after that she had again attempted to conquer the morphia, and had employed large doses of chloral against her sleeplessness. When the morphia was entirely suspended, dangerous nervous symptoms supervened. She was very excited; was often delirious; talked verses for hours; had tetaniform attacks with such intense prostration, that she was sometimes thought dead; had pains in every part of the body. Frequently she lost consciousness, but at other times she had intervals of perfect rest and clear-headedness, during which she earnestly requested to be taken to the institution.

During one of these lucid intervals she was brought to the institution on Dec. 7, 1877, in the evening. She spoke quite intelligently and sensibly about her condition, but within three hours she had a violent maniacal paroxysm. She dashed about the room, screamed, struck out, and could scarcely be held by four men. She was at the same time filthy in her habits. Next day she was quieter, but was frequently unconscious, and did not know where she was. In the course of December these attacks of excitement occurred repeatedly. At the same time her intelligence was impaired, her appetite bad, and large doses of chloral scarcely procured an hour's sleep. The urinary secretion was much diminished. She wasted considerably. Towards the beginning of 1878, during the first weeks

of the new year, she began to be quieter and gain strength. With this her mental condition quite changed. She was clearer, and generally recognised people; but she was highly crazed, had many hallucinations:—everything stank; there was a corpse in the room. She made repeated attempts at suicide; tried to set fire to her dress, and to strangle herself. After several weeks a state of apathy came on, which lasted till April. She scarcely spoke a word; frequently, however, she declaimed, lay almost constantly in bed, and took no interest in anything around her. In April she was again more active, but talked utter nonsense: "I will to the everlasting police; I have devoured my husband!" When out walking, she several times attempted suicide, trying to jump into a water cask. In May she was removed to another institution, which she left cured in May of this year (1879).

I saw her two months later. She was looking well, her mind had its normal clearness, and she said that she remembered scarcely anything of my institution. She remembered only one or two special circumstances.

In conclusion, I give the tables of the more important papers on Morphinism, so far as they are known to me.

Bernhuber.—Aertz. Intelligenzblatt, 1878.

Burkhardt.—Die Chronische Morphium-Vergiftung. Bonn, 1877.

Busey.—Philadelphia Medical Times, 1876.

Calvet.—Essai sur le Morphinisme. Thèse de Paris, 1876.

Erlenmeyer.—Die Recidive der Morphium-Sucht. Centralbl. f. Nervenheilkunde, 1879.

Fiedler.—Ueber den Missbrauch der Morphin-Injectionen. Zeitsch. für prakt. Medicin, 1874.

Kormann.—Deutsche med. Wochenschrift, 1877.

Krage.—Ueber Albuminuria und Glycosuria nach Morphium. Greifswald, 1878.

Kunz.—Die Morphiumsucht. Baier. Aertz. Intelligenzblatt, 1876.

Laehr.—Ueber Missbrauch mit Morphin-Injectionen. All. Zeitsch. für Psychiatrie, 1872.

Leidesdorf.—Die Morphiumsucht. Wien. med. Wochenschrift, 1876.

Levinstein.—Several papers in the Berl. klin. Wochenschrift, 1875, *et seq.* Die Morphiumsucht. Berlin, 1877.

Lewin.—Ueber Morphiumintoxicationen. Zeitsch. f. prakt. Medicin, 1874.

Martin.—The Opium Habit. Philad. Med. Times, 1874.

Mattison.—Philadelphia Med. and Surg. Rep. 1874. New York Med. Record, 1876.

Michel.—Ueber Morphiuminjectionen und Morphinismus. Würtemb. med. Correspondenzblatt, 1876.

Moinet.—On the Administration of Opium. Edin. Med. Journal, 1875.

Richter.—Casuistik zum Morphinismus. Berl. klin. Wochenschrift, 1876.

ILLUSTRATIONS OF DISEASES OF THE PONS VAROLII.

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THE special symptoms of acute lesions of the Pons Varolii may be briefly epitomised as follows:

1. The involvement, in unilateral lesions, of cranial nerves on the same side as the lesion.

2. The frequency of bilateral symptoms of paralysis in the cranial nerves and limbs. This depends on the fact that the motor tracts from the two sides are here still in contiguity, and on the circumstance that both are supplied from one vessel, the basilar artery, which is an occasional seat of morbid processes; degeneration, syphilitic disease, thrombosis, embolism.

3. The implication of the centres which are situated at either extremity of the pons, and which by their irritation and paralysis give rise to important localising symptoms. In the upper portion of the pons is the centre of origin of the oculo-motor nerves, irritation of which gives rise to the well-known contraction of the pupils, and damage to which may cause an equally important paralysis of the irides and ocular muscles. At the lower extremity of the pons are the centres which influence respiration, deglutition, convulsion, &c. Lesions which destroy these rarely leave time for diagnosis, but their irritation furnishes us with most important diagnostic indications.

4. The occurrence of hyperpyrexia, which appears to ensue from acute bilateral lesions irrespective of their character.

5. The occurrence of glycosuria, which probably is found only after the acute process is over, and has, in diagnosis, only a corroborative value.

Many of these symptoms are illustrated by the following examples of diseases of the pons Varolii,—embolism of the basilar artery, hæmorrhage, and senile softening.

CASE 1.—*Embolism of the basilar artery; general paralysis; myosis; hyperpyrexia.*

Wm. G., æt. 48, labourer, admitted into University College Hospital, Feb. 9, 1877. Rheumatic fever at the age of 28. For six months before admission he had suffered from shortness of breath, and for some weeks from bronchitis. He was a stout plethoric-looking man, presenting considerable dyspnœa, slight cyanosis, and œdema of the legs. Temperature normal. His lungs were emphysematous, and there were abundant bronchitic râles. Pulse 88, almost imperceptible, irregular and intermittent. Heart's action tumultuous; impulse diffused; dulness obscured by emphysema. First sound, rough; no murmur. Urine, sp. gr. 1024–30; albumen, ¼. Numerous hyaline, granular, and epitheliated casts.

On admission it was noted that the right pupil was larger than the left, both acting, however, to light. Two days after admission (on the 11th) he had a very severe attack of dyspnœa, without apparent cause. In the evening his pupils were contracted, but equal, and acting slightly to light. Patient was restless, wandering slightly; answering, however, when spoken to. This state continued during the night. At 7 A.M., on the 12th, he sank into the following condition. He lay on his back, breathing stertorously, skin hot, covered with perspiration. Pupils much contracted, equal, and insensible to light. He did not answer when spoken to, but could be slightly roused, opening his eyes when addressed in a loud voice. Eyelids habitually closed. Limbs not moved, and somewhat rigid; when pinched, an attempt was made to draw them away, but this seemed rather like a reflex than a voluntary action. At 10 A.M. his condition was nearly the same: when spoken to he still moved his head slightly and opened his eyes. Ophthalmoscopic appearances normal.

This state continued throughout the day. Early the next morning (3 A.M.) the coma had become deeper, and no sign of recognition could be elicited when patient was spoken to.

Pinching the arms still produced a slight movement. Pupils were contracted to a pin's point, and did not act at all to light. Swallowing impossible; temperature, 105·6; pulse, 130; respirations, 17. At 5 A.M. the temperature had risen to 109·2. A quarter of an hour later he was seized with a fit of coughing, became more cyanosed, and died in a few minutes.

The albumen in the urine lessened progressively from $\frac{1}{4}$ on the day of admission, to $\frac{1}{20}$, about, on the day before death.

Necropsy (30 hours after death).—Heart very large, weighing 23 oz. All parts flabby and dilated; the left ventricle especially, its wall scarcely $\frac{1}{2}$ inch in thickness. Orifices all dilated: mitral, $4\frac{1}{2}$ inches; aortic, $3\frac{1}{2}$ inches; tricuspid, $5\frac{1}{2}$ inches; pulmonary, 3 inches. A quantity of old membranous clot in the left ventricle, partly interlaced among the columnæ carneæ. Left auricular appendix distended with clot; from it there projected a mass of old clot, the size of a walnut, which had firm adhesions to the wall in front of the appendix. The muscular fibres of the wall of the left ventricle were very granular.

Kidneys large: left, 9 oz.; surface studded with small cysts; much fat in hilus; cortex narrow, consistence lessened; section opaque. Right: 7 oz., fewer cysts, firmer; cortex enlarged; epithelial cells very granular.

Liver and *lungs* congested.

Brain.—No lesion above the pons; arteries at the base fairly healthy. Vertebrals and basilar free from atheroma. The anterior half of the basilar distended with clot, reaching forwards as far as the anterior cerebellar arteries. The anterior portion of the clot was pale, and distended the vessel more than the other part of the clot, which was dark, recently-formed coagulum. The anterior portion was evidently an embolon, and resembled perfectly the old clot in the left auricle. Both superior cerebellar arteries contained fluid blood, except that the first quarter of an inch of the right contained a coagulum continuous with that in the basilar. Both posterior cerebrals also contained fluid blood, although clot extended into the commencement of the right, which was very much smaller than the left—about one-fourth of the size—the right posterior communicating artery being correspondingly large. The pos-

terior half of the basilar, and the vertebrals, contained fluid blood.

The lower half of the floor of the fourth ventricle presented no vascular engorgement, only one or two vessels of the smallest size being visible. In the upper half a large vessel on each side was enormously distended with blood, and the grey matter had a redder tint than normal. The consistence of the upper half of the pons was slightly diminished, but there was no actual softening. (Being intended for a museum preparation it was not further examined.) The cerebellum was healthy.

The case was a typical one of embolism of the basilar general paralysis, with persistence of quasi-voluntary reflex movements, peculiar incomplete coma, irritation of the nucleus of the third nerve (or pupillary centre) leading to the extreme myosis, and with hyperpyrexia.

It is remarkable that some authorities doubt the occurrence of embolism of the basilar artery. Nothnagel, in his recent most admirable work on cerebral localisation,[1] says, "Acute embolisms of the basilar artery, limited in effect to the pons, do not occur. The reason for this is clear, on consideration of the anatomical relations of the vascular arrangement of the vertebral arteries, which renders the entrance of a plug into them extremely difficult, and on consideration that these vessels have a narrower lumen than the basilar artery which arises from them. If thus a plug should enter the vertebral artery, it must remain arrested therein, and it can, at most, cause only a gradual thrombotic occlusion of the basilar by the deposit of clot."

This reasoning is, I think, scarcely conclusive. The passage of a plug into a vertebral does undoubtedly occur now and then. The posterior cerebral artery may be plugged by an embolon. The difference in size between the vertebral and posterior cerebral arteries is considerable. A plug may be small enough to pass through the vertebral and yet too large to pass into the posterior cerebral. In this case it must be arrested at the anterior extremity of the basilar,

[1] 'Topische Diagnostik der Gehirnkrankheiten:' Berlin, 1879.

and whether its effects are limited to the pons or not will depend on the size of the posterior communicating arteries. As regards embolic processes, the basilar is an indifferent continuation of either vertebral, and the anterior portion of the basilar is the necessary place of arrest for a clot just small enough to pass through the vertebral. Probably Nothnagel's opinion is intended to refer to plugging of the course of the basilar, not of its anterior extremity. But many embolic plugs are much longer, in their longest dimension, than is the transverse diameter of arteries through which they pass. Such a plug may be washed along the vertebral, and plug a considerable extent of the anterior portion of the basilar, and even under the pressure of the blood behind may distend the vessel so as to give the impression of a plug which could not have passed through the vertebral.

Thrombosis in the basilar artery, the consequence of arterial disease, atheromatous or syphilitic, is however unquestionably a more frequent lesion than embolism. It probably existed in a case (seen with Dr. Vereker Bindon) in which the disturbance of respiration was very striking. The patient was a stout woman, aged 56, who had had several slight rheumatic or gouty attacks. After exposure to cold she became ill, complaining of sciatic and lumbar pains, and some left-sided pleurisy was found three days later. Her urine was free from albumen. In the afternoon she complained of ringing in the ears, suddenly became slightly delirious, and then partially unconscious; pupils slightly contracted, equal, and still sensitive. Respiration became embarrassed, without anything being discoverable in the chest to account for it, the breathing, at intervals of about twenty minutes, becoming slower and slower, until it ceased; but on artificial respiration being adopted, it gradually recommenced and gained in strength for about ten minutes, when it again went through the same process. At 10 P.M. she was lying on her back, moving the head uneasily from side to side, and at times opening her eyes, but not the least evidence of consciousness could be elicited. The pupils were of medium size, equal, not acting on the least to light. The arms were flexed at the elbow and rigid, with constant

jerking, half-co-ordinated, but evidently involuntary, movements of the hands. The legs were motionless. Respiration was very irregular, frequently stopping for a few seconds and then going again. Skin perspiring, temperature 102°. Pulse 120. The intermitting respiration continued through the night and next morning became extremely irregular, often ceasing for a minute at a time. She died next day at noon. There was no post-mortem.

In this case the bilateral symptoms, the convulsive movements in the arms, the inaction of the pupils, in contrast with the apparently incomplete degree of coma, the initial tinnitus, and lastly the extreme disturbance of respiration, point strongly to a lesion of the pons, probably thrombosis of the basilar artery, due to the blood state.

Degeneration of the basilar artery usually produces its effects by leading to thrombosis within it, but may lead to fatal results without this, by obliterating the branches which are given off to supply the pons. This occurred in a patient under my care several years ago, of whose case I possess only a very brief record. He was a man about 50 years of age, who had had a transient attack of left hemiplegia six months before he came under treatment, and two weeks before being seen he had a fresh attack, also on the left side, coming on without loss of consciousness, and reaching its maximum in three days. On admission into hospital there was complete paralysis of the left arm and leg, and moderate paralysis of the face without loss of sensation. Two days subsequently the right arm and leg also became weak, and the weakness increased during the next two days. Respiration became frequent (45 per minute), the chest full of râles, the skin perspiring. He died three days after the commencement of the right-sided weakness. His skin before death was said to have become extremely hot. The post-mortem revealed no lesion in the hemispheres. The basilar artery was extremely atheromatous, and there was an area of softening on the right side of the pons near the middle.

In another case of thrombosis in the basilar from syphilitic disease, lately under my observation, the temperature rose before death to 105°. A similar temperature was observed in the next case of rapidly fatal hæmorrhage into the pons.

CASE 2.—*Hæmorrhage into the Pons Varolii: oculo-plegia; respiratory disturbance; convulsion in legs; hyperpyrexia; death within an hour.*

A man, apparently about 60 years of age, was brought to University College Hospital about a quarter of an hour after he had fallen unconscious in a public-house. He then presented no sign of consciousness, eyes partly open, pupils medium size and absolutely insensible to light. Conjunctiva insensitive to touch. Pulse weak and regular, not infrequent. Breathing was not stertorous, but was markedly irregular and interrupted: three or four respirations were taken, and then a long pause ensued, during which there was some venous congestion of the face. At the end of each breathing period, when the chest was full of air, there was a forcible respiration, very like a sneeze, which jerked his head off the couch for some inches. Then followed the pause. The arms were motionless and flaccid. The lower limbs and muscles of the abdomen were affected with "convulsive tremblings."

I saw the patient immediately after his admission into the ward. The peculiar sneeze-like spasm of respiration had ceased: his breathing being still very irregular. The convulsive movements in the legs continued—constant, quick, jerking movements, equal and synchronous in the two. After about half an hour the arms also became rigid and convulsed. His temperature, when first seen, appeared normal, but soon after admission into the ward it was 103°. He died, asphyxiated, three-quarters of an hour after admission, and about an hour after the seizure. At death the temperature was 105°·4.

At the necropsy a large clot was found to occupy the pons Varolii, being most extensive in its upper and anterior portion, the upper (posterior) half of the pons being destroyed and the anterior (inferior) portion considerably damaged. The clot had burst through into the fourth ventricle, which was filled with blood—the upper half of the floor being completely broken up. The destruction ceased below at the auditory striæ. It extended to the surface in the middle of the pons, separating the superficial fibres midway between the right fifth and sixth nerves. It extended upwards beneath the corpora quadrigemina,

where it reached backwards along the middle line almost to the surface, completely destroying the nuclei of the third nerves. The inner side of the left crus was torn by the

Fig. 1.

Section through the crura cerebri (*c c*) just above the pons: the space between them at the upper part (below in the figure) is occupied by hæmorrhage, which extends almost to the under surface of the pons (above in the figure), and has extensively damaged the corpora quadrigemina and destroyed the nuclei of the third nerves. (*From a sketch by Mr. Neale.*)

hæmorrhage, and the right crus in less extent. The hæmorrhage had extended into the right crus cerebelli, but not quite so far as the corpus dentatum. The third ventricle was filled with clot, and there was fluid blood in each lateral ventricle. There was a little blood at the base beneath the arachnoid. Very little atheroma of the vessels.

The left lung was intensely congested. The lower lobe appeared to be a mass of blood. The substance of the lung was black, but of normal consistence, and crepitant. The upper lobe was loaded with fluid but not congested. The right lung showed only some old changes. The left kidney was enlarged and slightly indurated: the right atrophied, in consequence of an impacted calculus in the pelvis, which had caused hydronephrosis. In the stomach, over the left third, the mucous membrane was covered with extravasations, small, recent, arranged in an arborescent form.

The case illustrates many points of interest in the diagnosis of these diseases. The nuclei of the nerves moving the eyeball and the pupil were destroyed. Hence both eyeballs and pupils were motionless, and the pin-point con-

traction, which exists when the lesion merely irritates the nuclei of the third, was absent.

The respiratory centre situated just below the lesion was irritated and disturbed, and hence the remarkable respiratory symptoms.

The elevation of temperature in disease in this situation is of interest in its diagnostic as well as its physiological relations. The diagnosis of hæmorrhage into the pons was doubted, at the bedside, by some of the students, on the ground that hæmorrhage causes an initial depression of temperature in all cases. In some authorities the statement is certainly made too absolutely; the fact of the special effect of lesions on the temperature of the pons, which is independent of the form of lesion, being overlooked.

The extreme congestion of the left lung and the extravasations in the left end of the stomach may possibly have been due to the irritation of the nucleus of the pneumogastric.

CASE 3.—*Softening of the Pons Varolii; hemiplegia; subsequently opposed facial palsy; diplegic spasm; glycosuria.*

Robert J., five months before being seen, was attacked with right hemiplegia: there remained considerable weakness of the right arm and right leg: very little paralysis of the right side of the face. On December 13th, four months after the onset of the hemiplegia, after riding in a wet cab, he felt pain in the left jaw, which became so intense as to prevent him from sleeping: it radiated from behind and below the left ear over the side and front of the head, and over the left side of the face. The intense pain lasted three or four hours. On waking next morning the left side of the face was completely paralysed in all parts. The pain continued until he was seen on January 3rd. The paralysis of the face had improved slightly, but was still nearly complete. Faradic irritability 4 or 5 cm. (Stöhrer sec. current) less than right side; voltaic irritability four cells greater. The application of the voltaic current to the right side, especially to the point of exit of the second division of the fifth, or to the forehead, the other pole being placed in front of the ear, excited contractions in the muscles of the left side of the face, especially in the lev. labii sup., and to a less

extent in the zygomatici. They occurred on closing the circuit, and with very feeble currents (four cells). The same current which, passed from the ear to the forehead on the right side, excited a strong contraction in the muscles moving the angle of the mouth on the left side, caused no contraction in these muscles when applied to the left side of the chin. Further examination revealed distinct hyperæsthesia of the second branch of the right fifth nerve: pressure on, or pinching the skin causing much more uneasiness than on the left side.

On February 8th his urine, which had not been before examined, was found to contain a distinct trace of albumen, and a considerable quantity of sugar, ascertained next day to amount to 1800 grains in the twenty-four hours' urine, on the 12th to 2835 grains, and on the 14th to 1800 grains; the quantity of urine varying from 90 to 110 fl. oz. He was conscious of an increased flow of urine, and was confident that this had come on since the attack of hemiplegia. On strict diabetic diet the urine fell, in the course of a week, to 45 oz., and the sugar to a trace too small to estimate quantitatively. During the next two months the urine remained the same in quantity, and the trace of sugar persisted. The facial paralysis improved slowly. On May 12th he died suddenly—in a moment.

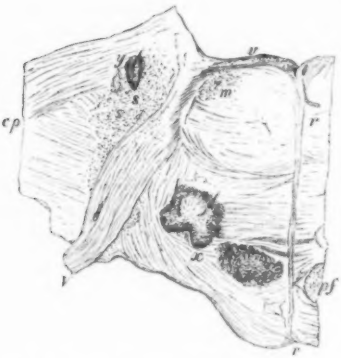
The necropsy showed a fusiform dilatation of the arch of the aorta, as far as the left subclavian, its lumen being about four times the normal. The heart healthy for age (11 oz.). The kidneys were large (8 and 11 oz.), opaque and indurated.

The brain above the pons Varolii showed no evidence of disease. The arteries of the base were very atheromatous, but nowhere occluded.

The pons Varolii, medulla, and spinal cord were carefully examined after hardening. The pons, just behind the origin of the crura cerebri, was equal in size on the two sides; but a little lower, opposite the upper portion of the middle peduncle of the cerebellum, there was a depression in the convexity of the left side; and opposite this, in the middle of the pons, was an irregular area of softening, situated in the pyramidal fibres, opposite and a little below the origin of the fifth nerve. Above, the softening was in two foci (*x*) (Fig. 2, p. 476), in one of which was a cavity, the other being occupied by

granular débris, but a little lower these two had blended into one of rather smaller size. The fibres of the fifth nerve and of the facial nerve were both apparently undamaged; the

Fig. 2.



Section through the left half of the pons Varolii at level of exit of fifth nerve: *v*, floor of fourth ventricle; *c p*, middle peduncle of cerebellum divided; *r r*, raphe of pons; *V*, fifth nerve at its exit; the fibres are seen ascending into the substance of the pons; *s*, upper part of the sensory nucleus, in which are two foci of softening, *y*; *m*, highest part of motor nucleus of fifth; *p f*, pyramidal fibres on right side ascending through the pons from anterior pyramid; *x*, two foci of softening, destroying all the ascending pyramidal fibres on the left side.

lesion, in its lower part, was close to the fibres of the facial. At the level figured, the sensory nucleus of the fifth (*s*) presented two small foci of softening (*y*). The anterior pyramid on that side was completely degenerated, and the degeneration could be traced through the decussation of the pyramids to the opposite lateral column of the medulla in the usual course.

Remarks.—The early notes of this case are imperfect, although the patient was very carefully examined, the symptoms present being those of disease of the corpus striatum with a small amount of paralysis of the face. The occurrence of facial paralysis led to a careful investigation with electricity, which revealed the peculiar diplegic contractions. From these disease of the pons was suspected, which the existence of glycosuria supported and the autopsy confirmed.

Although the autopsy did not reveal any actual lesion of the facial nerve within the pons, it seems highly probable that its paralysis was due to inflammation extending from the adjacent focus of softening, and that this extension and the lesion in the nucleus of the fifth occurred simultaneously. To the latter, the intense pain in the region supplied by the fifth may have been due.

The contractions in the left side of the face, when the right side was galvanised, can only have been produced by one of two modes. Either they were the result of direct stimulation of the very sensitive muscle by the irradiation of the voltaic current, or they were reflex. It seems impossible that they can have been due to the wide diffusion of the current. A current of four cells applied in the way described, could scarcely cause contractions in the muscles of the other side of the nose, especially when it is considered that a much stronger current, applied below the angle of the mouth on the left side, did not lead to the contraction of the levator labii superioris, &c., which followed the application to the right side.

The cause of the diplegic reflex was probably an undue sensitiveness of the fifth nerves, perhaps due to the lesion found in the nucleus of the left fifth, reacting on the right nucleus.

The occurrence of glycosuria in diseases of the pons which do not actually involve the "diabetic point" has been before observed. In this case the fact that the patient had observed the increased flow of urine only since the hemiplegia made it highly probable that the diabetes was a sequel to the paralysis. It is not necessary, in explaining the phenomenon, to assume that the focus of disease is related to the glycosuric processes. The vascular disturbance secondary to the obstruction which caused the softening would, no doubt, be very considerable, and may have extended to the "diabetic point."

ON THE ORIGIN OF TETANUS.

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TETANUS is one of those few remaining, striking, and fatal diseases, which deeply interest the public mind, as having so long resisted the art of medicine, and baffled its inquiries. Not, indeed, that cases of tetanus are invariably fatal. From time to time well-authenticated cures are reported in the papers. But there is as yet no received theory as to the nature of the disease; there is no acknowledged line of treatment; and the number of deaths in acute cases is out of proportion greater than in most other diseases. Acute tetanus is even more fatal than cholera. Poland gives the mortality of all cases, as seven deaths in eight and a-half cases.

How comes it that we have advanced so little in this direction? Probably because there are still different, and even contradictory, views held and taught as to the very nature of the disease. There are humoral pathologists—a good number—who look upon tetanus as a species of blood-poison. Others trace it to inflammation of the nerves and spinal cord; while the majority, confronted with the dual origin of the disease, expressed by its classification into Traumatic or Idiopathic, consider, in fact, that it may arise either one way or the other. Of late, however, there is a growing tendency on the part of writers on tetanus to give up all of the foregoing theories as to the origin of the disease as lacking proof, and being inconsistent with many well-known facts. But, so far, nothing very definite has been put forward in substitution.

And first as to its humoral origin, which finds expression so often in the term Idiopathic, as distinct from the Traumatic

form of the disease. What proof have we of it? As a rule, perhaps, so-called idiopathic cases are milder than cases arising from injury; but the exceptions to this rule, both ways, are so numerous, that no argument can be founded on this. Many idiopathic cases are very acute; idiopathic tetanus is often fatal. Many traumatic cases, on the other hand, are very mild, and do not end fatally. The fact is that tetanus has grades of severity, like other diseases; acute cases, however arising, are generally fatal; and chronic cases, however arising, are generally not fatal; and the division into acute and chronic is a good and practical one, but there is no positive proof of its idiopathic origin in any single case.

It is easy to deny the humoral or idiopathic origin of tetanus. I must use the term idiopathic in this paper, to make myself clear, since it is a part of the language of the subject; but I do so with the protest that the word is in this connection a mere negative. It really means nothing more than that the cause of the disease is not apparently traumatic. It has, besides, the great practical drawback of suggesting a humoral origin where none has actually been found.

No one, as far as I know, denies that traumatism is one cause of this disease. There are so many unequivocal proofs of it on record, that it would be rash to do so. I will give one out of many for the sake of illustration, premising that the case is an unusual one. Sir T. Watson relates that Professor Robinson, of Edinburgh, was once at table when a negro servant lacerated his thumb by the fracture of a china dish. He was seized with convulsions almost instantly, and died with tetanic symptoms in a quarter of an hour. This case, it will readily be admitted, leaves no opening for humoral pathology. There are many cases which go nearly as far to demonstrate the traumatic origin of the disease. I think I may, to save space, take so much for granted, that tetanus does often arise from injury. It is generally so admitted, and all cases of tetanus following an injury are classed as traumatic. This leaves a large residue of cases to be dealt with, in which no injury is apparent, and which are for that reason called idiopathic, and for no better reason. I might here suggest that the traumatic origin of tetanus is often so insignificant as to be overlooked.

Sir T. Watson mentions cases of tetanus following the bite of a sparrow; the stroke of a whip-lash, in which the skin was not broken; a fish-bone in the fauces; a blow of a stick on the hand. Sir W. Ferguson, the scratch of a pin. Bryant, a slight contusion. Erichsen, a child falling on its back; no breach of surface. And doubtless some, so-called, idiopathic cases have grown out of some such trivial injuries. But the difficulty of explanation must be fully considered, and fairly met. What is the difficulty? That a number of cases of tetanus occur without the intervention of a wound or injury. And, what if the intervention of a surgical wound or injury be an accident of the disease, and not an essential? I am far from considering a surgical injury as the invariable starting-point of tetanus. On the contrary I would give up traumatic as well as idiopathic, looking upon both members of the classification as misleading. I will try to show that tetanus is a single disease; that it begins in persistent peripheral nerve-irritation, and ends in organic molecular, and functional disturbance of the medulla. This peripheral nerve-irritation need not, however, be traumatic.

I will divide the subject into five propositions. 1st. Peripheral nerve-irritation is a cause of tetanus. 2nd. Peripheral nerve-irritation is present in all cases of the disease. 3rd. It produces eventually the group of symptoms known as tetanus. 4th. It explains the facts of the morbid anatomy of the disease. 5th. It guides the treatment of the disease, and is proved by its success.

Three cases may be cited to prove that peripheral nerve-irritation is a cause of the disease. 1st. Dr. Murray, in the eleventh volume of the 'Medical Gazette,' records the case of a midshipman, who whilst on night-watch trod upon a rusty nail, which pierced his left foot. Soon after, his foot and leg felt numb and peculiar, and the next morning symptoms of acute tetanus set in. Dr. Murray at once divided the left posterior tibial nerve with a stroke of his scalpel. The patient felt immediate relief; his mouth opened; the disease subsided, and he was well in two or three days. Now the way in which I read this, and the only way in which I can understand it, is, that a peripheral branch of the left posterior tibial nerve was

injured, or irritated by the nail or wound, and that the irritation was propagated along the nerve to the medulla, and gave rise to tetanus; and that, upon the division of the nerve, the irritation ceased to flow up to the medulla, and the reflex symptoms (*i.e.* tetanus), naturally subsided. Volkman has pointed out that irritation of a nervous centre, through a centripetal nerve, causes contractions, which last after the withdrawal of the stimulus. 2nd. A similar case rests upon the evidence of Baron Larrey. Tetanus followed an injury to the supraorbital nerve, and was directly cured by division of the nerve. This case is mentioned in most works on surgery. The same remarks apply to it as to Dr. Murray's case. 3rd. Bryant reports that Mr. Key, in 1845, amputated a leg for tetanus, which had followed on an unreduced dislocation of the astragalus. The symptoms disappeared at once after the operation. On dissection, the posterior tibial nerve was found to have been put violently on the stretch by the projecting astragalus. In this case there is no wound; septic influence is excluded; a peripheral nerve, the posterior tibial again, is kept violently on the stretch; irritation is propagated to the medulla; the train of reflex phenomena, known as tetanus, ensues; the source of irritation is cut off by the knife; the symptoms of reflex irritation cease immediately. Nothing could be plainer. I take these three cases as proving that peripheral nerve-irritation is a cause of the disease—the first step in this inquiry.

There are very many cases mentioned in surgical works, which go to strengthen the above proposition. Erichsen gives five or six cases, in which post-mortem examination revealed laceration, puncture, or other obvious injury of the nerve of the part. He seems to consider peripheral nerve-irritation as the starting-point of the disease in traumatic cases. Many other eminent surgeons agree in this. No one, as far as I know, has ever brought forward proof that any other cause exists. On the principle that like effects from like causes arise, this cause may safely be extended to all traumatic cases. Indeed, peripheral nerve-irritation is an essential feature of traumatism, and is included in the definition. Proposition the first being proved above, the following may be taken as a

corollary. Traumatic tetanus arises from peripheral nerve-irritation.

Let us see now what bearing this has upon so-called idiopathic tetanus. First, I must recapitulate that there is not an iota of direct proof that tetanus is ever idiopathic, whatever the word may mean. The term is here used simply in a negative sense. It is the negation of injury. If we accept peripheral nerve-irritation as the source of one form of tetanus, we must exclude it from the other, to justify the classification by negation. The question then arises—Is, or is not, peripheral nerve-irritation present in idiopathic tetanus? This question is embraced in the second proposition: viz. peripheral nerve-irritation is always present, which I will now endeavour to prove.

The great bulk of idiopathic cases are, in point of fact, either puerperal, menstrual, or the result of worms. They are put down as such in hospital records; this is well known, and easily proved. Indeed, there are authors—as MM. Laurent and Lombard—who go so far as to say that tetanus, even when traumatic, is almost always caused by worms. So common is this association of worms with tetanus at Madras, that santonin is a stock remedy in idiopathic tetanus. Again, puerperal and menstrual cases are exceedingly common. Looking over the records of the hospital, which I am now in charge of—the Women and Children's Hospital, Madras—I find that all the idiopathic cases which have been recorded in detail, including uterine and vaginal diseases, are either puerperal or menstrual, or the result of worms. Dr. King, formerly in charge of the Triplicane Hospital, Madras, found that out of 224 cases of tetanus treated in that hospital, 111 were males, 113 females. He makes the remark: "These numbers are nearly equal, showing that menstrual and puerperal predisposing causes in the one sex, about compensate the greater liability of the other to mechanical injury." In these acknowledged forms of idiopathic tetanus, peripheral nerve-irritation is undeniably at work. I have examined the gut (post-mortem) in cases the result of worms, and have found chronic inflammation, ulceration, worm-burrowing, and other signs of considerable local irritation. I have examined the utero-

vaginal tract in puerperal and menstrual cases, and have found congestion, erosion, ulceration, clots, and other signs of local irritation. To deny peripheral nerve-irritation in these cases, would be to deny that the parts concerned are supplied with nerves. It follows that in a large class, the greater part of idiopathic cases, peripheral nerve-irritation is at work. Now, if peripheral nerve-irritation at the surface causes tetanus, as has been proved to be the case, it is highly probable that it acts in the same way beneath the surface; that is, in the interior of the body.

There is but one step more in this inquiry, and that is to include all idiopathic cases. We must infer from the foregoing that any source of peripheral nerve-irritation within the body is capable of producing tetanus. Now, what are the possible sources of nerve-irritation within the body? Their name is legion. Parasites of all sorts, ulcers, concretions, tubercles, vomicæ, morbid growths, &c., diseases of the lungs, liver, bladder, and other organs. In a word, any disease, anywhere in the body, is a source of nerve-irritation; and that case of idiopathic tetanus from which all such sources could be excluded must be absolutely free from disease. I cannot imagine that such a case has ever existed. Bryant says: "The late Mr. Wilson King, of Guy's, is stated by Poland to have been in the habit of remarking at the post-mortem table, whenever there was an examination of a case of death from tetanus, 'Gentlemen, we will now proceed to give you demonstration of a case of healthy anatomy; for there will be no visible morbid appearance otherwise than congestion of the organs in various degrees, which are owing to accidental circumstances.'" What did he mean by congestion of the organs "owing to accidental circumstances"? what organs, and what circumstances? Seeing that his remarks apply to traumatic cases, and perhaps to them chiefly, the organs referred to may be those of the nervous system, in which case the inference is clear. Anyhow, supposing his post-mortem examinations were more than usually searching, a vast number of sources of peripheral nerve-irritation in the organs, vessels, nerves, muscles, bones, and tissues generally, would, if present, escape detection. So that his remarks do

not in the least affect my second proposition that this cause, viz. peripheral nerve-irritation, is always present. This post-mortem question will be fully discussed under the heading of Proposition the 4th.

I now come to the third part of this inquiry: Does nerve-irritation give rise to the group of symptoms known as tetanus? If we were to reason from the known symptoms of the disease back to their hidden cause, it is precisely nerve-irritation that we would expect to find at the bottom of every case. The symptoms of tetanus are those of constant irritation of the excito-motor system, with great exaltation of the reflex function of the cord and medulla. It is characterised by muscular spasms, tonic and clonic, the latter brought on by the least noise or motion, the slightest stimulus to the peripheral nerves. The brain is clear and unaffected. There is often no fever, no constitutional disturbance of any kind. Physiologists know that if one of the spinal nerves of a headless turtle be irritated, contraction of the muscles follows on both sides of the body, both above and below the roots of the nerve operated upon. The irritation goes along the afferent nerve to the medulla, the centre of reflex irritability and seat of decussation of motor nerve fibres, from whence the impulse of motion is sent through the efferent nerves to any muscle in the body.

So wide-spread are the connections of the medulla that its afferent nerves are involved in every disease or injury within as well as without the body. Tetanus can be produced artificially in a headless frog, which has been rendered excitable by the injection of opium into the blood, by irritating the peripheral nerves (Kirke). Similarly with strychnine.

The pathology of tetanus would seem to be this:—1st. Continued irritation of the peripheral extremity of a nerve or nervus nervorum, through injury or disease of some sort. 2nd. Continued irritation of the medulla, with tonic reflex spasm, risus sardonicus, and trismus. 3rd. The unbroken cycle of continued irritation leads to morbid molecular changes, with violent disturbance of the reflex function and paroxysmal tetanus, on the least excitation, as a current of air.

Clinical facts support the idea that the medulla is early

affected. Nothing is more remarkable in tetanus than that it always begins with trismus, risus sardonicus, and stiffening of the sterno-mastoid muscles. These symptoms clearly denote irritation of the medulla, inasmuch as these muscles are supplied by nerves, the inferior maxillary division of the 5th, portio dura of the 7th, and spinal accessory, all of which arise from the medulla. Why these parts should be the first affected, is another question. It may be because they are nearest to the medulla, which thus expends its purposeless reflex force by the shortest route. Thus, for instance, if a drop of boiling water falls upon the hand, it is involuntarily withdrawn by a well-meant reflex act quicker than thought. But if the hand be well scalded, there is no meaning in the continued peripheral nerve-irritation which results, and which is a common cause of tetanus. Here we may suppose that the reflex irritation is transferred by the medulla to the nearest muscles by the shortest route, to expend the agony as it were, for nature perceives that to move the hand would now but increase the pain and reflex irritation. To avoid drawing out further the pathology of tetanus, which would be a digression in this place, I will conclude this branch of the subject by saying that Proposition the 3rd has been demonstrated experimentally. Peripheral nerve-irritation produces eventually the group of symptoms known as tetanus.

Fourth.—Does peripheral nerve-irritation, taken with the disturbance of the excito-motor function which it causes, account for the actual facts of morbid anatomy as found in post-mortem examination of patients dying of tetanus? Reflex irritation is a dynamical phenomenon. In its most distorted form we need not necessarily expect to find its track, post-mortem, as an objective morbid state. There is a strong antecedent probability that in some cases—acute cases of some duration—molecular changes do take place; but then such changes may exist with an undisturbed appearance of parts even with the aid of the microscope. Now there are many cases of tetanus on record in which no morbid change of any kind could be detected in the cord post-mortem. Bryant, Billroth, Nothnagel, and others—men who have the reputation of being careful observers—testify to this. Billroth says, "My

investigations of the spinal cord and nerves in tetanus have, as yet, given negative results only." This is not surprising, considering the nature of the disease. A great number of observers of equal weight, however, state that they have found marked changes, some in the cord, others in the medulla, others again in the floor of the 4th ventricle; and in traumatic cases inflammatory changes have been found in the peripheral nerves implicated. Under the microscope, parts apparently sound have been shown to have undergone change. At a late meeting of the Pathological Society, London, April 29, 1879, Dr. Ross showed microscopic sections of the cord after tetanus. He found dilatation and plugging of vessels, migration of leucocytes and of a granular material round the vessels, chiefly in the grey substance of the cord and in the grey nuclei in the floor of the 4th ventricle. Besides these inflammatory changes, he found the cells, especially the median group of the anterior horn of the columns, injured. Lockhart-Clarke, Erichsen, Allbutt, Gowers, Coats, Dickinson, and Benedikt have described similar changes. Aitken speaks of increase of specific weight; Rokitansky and others of a development of fresh connective tissue. In two so-called idiopathic cases, one puerperal, the other the result of worms, which I examined lately, the following changes were found:—bright-red vascular patches on the dura mater over the medulla, colour persistent after repeated washings; vessels of the pia mater much engorged; arachnoid opaque and of a brownish tint, and excess of serous fluid. These changes are not in any way inconsistent with the proposition stated above; for although their absence does not militate against it, their presence is certainly more in accordance with what physiology would lead us to expect. Exalted function is always accompanied by hyperæmia. Increase of work necessitates increase of blood supply, and the line of demarcation between healthy plethora and morbid engorgement is often a very fine one and easily overstepped. Witness the condition of the breast at full time, itself a reflex phenomenon dependent on the condition of the uterus. We would expect to find post-mortem evidence in some cases of congestion, in others of inflammation and molecular disintegration; but we could not, from the nature of the

disease, expect to find such changes always, and this is exactly what happens. The length of time the disease has existed, and its intensity, should, moreover, influence the result. A mild case ending fatally in a very debilitated subject would not probably yield such marked changes as a severe case in a robust subject. The negro, mentioned by Dr. Robertson, who died in a quarter of an hour from spasm of the glottis (?), would not present any change at all. According to the view of the subject herein argued, in the first stage of the disease, that of continued nerve-irritation reacting upon the medulla and originating tonic spasms, up to a certain point there is no morbid change, and consequently there would be no evidence of disease. At this period division of the afferent nerve, by cutting off the source of irritation, arrests the symptoms. Afterwards, continued irritation exciting the reflex function to an inordinate degree, causes hyperæmia with molecular changes, and frequently inflammation. Here there may or may not be visible change, but there will be hidden molecular changes. Pathological facts fit in exactly with these views. Sometimes no changes are found, sometimes microscopical changes, and sometimes evidences of inflammation of the medulla and upper part of the cord. Proposition the 4th—peripheral nerve-irritation explains the facts of the morbid anatomy of the disease.

Here I must digress to say that I once thought that the disease itself was a species of neuritis ascendens; but the absence of post-mortem evidence of inflammation in many fatal traumatic cases, both in the peripheral nerves implicated and in the cord, makes this view untenable. Professor Nothnagel of Jena, who has specially studied this view of the question (*vide* New Sydenham Society's Second Series, 1877), has come to the conclusion that it cannot be proved either by clinical or pathological evidence, and he is forced to the conclusion that tetanus is due to an increase of nerve-irritability, possibly resulting from molecular changes beyond demonstration.

There remains the fifth proposition, that the peripheral nerve-irritation view of tetanus is borne out by its treatment. I have already brought forward three cases, in two of which, section of the nerve, and in one, amputation, cured the disease,

as a proof of its origin in nerve-irritation. These three cases are of equal value in proving any present proposition. Later on I stated that this treatment, by the knife, is only efficient up to a certain point; that when molecular changes take place in the medulla it is useless. Division of the nerve in the second stage may help, but does not cure the disease. Excision of the offending part does not affect it. Amputation of the limb is of no avail. All these have been tried and have failed signally. Something more is wanted—some powerful wrench to the nervous system that will alter the morbid molecular arrangement of the parts, or act as a revulsive or counter-irritant. Stretching a large nerve trunk and making traction on the cord, for instance, may prove of use. Well, this has been tried. The sciatic nerve has been stretched, and with marked success. I have seen it abolish clonic spasm for hours, in some acute traumatic cases, arising from injury to the limb operated upon. It may be said that this also acts by stopping the current of peripheral nerve-irritation. I think not. I think its action is upon the molecules of the spinal marrow or as a revulsive for this reason. The conductivity of the nerve itself is apparently unchanged. Sensation, nutrition, and motion, remain unimpaired in the limb operated on. Again, Prof. Annandale of Edinburgh lately stretched the spinal accessory nerve for spasmodic wry neck, without effect; whereas a cure immediately followed subsequent division of the nerve. In some cases of leprosy, and indeed in other special cases, the nutrition of the hand is improved by stretching the ulnar nerve. In 1878, Baum of Danzig cured a mimic spasm by stretching the facial nerve. Clearly then the nerve still conducts, but the mechanical violence gives rise to mechanical changes in the nerve centres or something of the kind, whereby reflex excitement is for a time subdued (?). If this view of the action of nerve-stretching is correct, it should act more or less well in all cases, whether puerperal or the result of worms, or what not, according as the afferent nerve implicated entered low down or high up the cord. I have made trial of this crucial test in a so-called idiopathic case (puerperal); but the case was hardly acute enough for my purpose. There was complete trismus, compelling rectal

alimentation; but medicines were at times given by the mouth and swallowed. I saw no spasms of opisthotonos, but the attendants, apothecary and nurse, said that they had. I stretched the right sciatic nerve with a tension of 10 lbs.; there were certainly no spasms after the operation, and the patient ultimately recovered under the influence of chloral. The case goes to prove my proposition, but not so far as if it had been more acute. I will not venture to infer anything, however, from the result of this single case, the more especially as, since writing then, I have had a case which seems to contradict it. A girl aged eight years was seized with acute tetanus ten days after having the cartilage of her ear pierced for an ornament. I stretched the left sciatic up to 12 lbs. on the day of admission, and, with chloral, she improved very much; but next day the clonic spasms were worse than ever. Seeing this, I opened the wound and stretched the sciatic again, but without the least effect. The girl died that night. Here failure may have been due to the fact that the sciatic nerve is a long way off from the cervical plexus, whose branches were irritated, but this is a separate question and an obscure one. It would have been better in this case to have stretched the cervical plexus. As regards treatment, the fact of greatest significance perhaps is, that whenever we look, we find men of all shades of opinion treating the disease as if it arose from peripheral-nerve irritation and had its climax in distortion of the reflex function of the medulla. Billroth himself originated nerve-stretching in traumatic cases. Surgeons almost invariably amputate fingers and toes when tetanus follows on their injury. And for constitutional treatment, the drugs used are just those which are known to diminish the reflex irritability of the cord, such as chloral, atropia, calabar bean, bromide of potassium, chloroform, alchohol, opium, and the like. And moreover, such drugs have justified their use in this disease. There is hardly one of them which has not been credited with a number of well-authenticated cures. They have each their special advocates; but no one doubts that all in turn have from time to time successfully combated the disease. The ice-bag to the spine, leeches, blisters, and plasters, to the same, all testify to the universal opinion, acted

upon, if not avowed, that tetanus is a disturbance-disease of the reflex function of the cord. I need hardly dwell further on this point, the 5th proposition, It (the peripheral nerve-irritation theory) guides the treatment of the disease and is proved by its success.

I will now consider the force of these five propositions taken together, as evidence that peripheral nerve-irritation is the cause of tetanus.

If a number of murders took place in different parts of England, and all were of the same kind—by strangulation, for instance—and one man was always found present, no matter when the murder took place, strong suspicions would rest upon him, even though there was no direct proof of his guilt. But if subsequently this man was proved to be a Thugge, a man who had been convicted of at least three murders, and that by strangulation, a jury would surely find that he had been guilty of all. I am imagining a very strong case, one in which there is no evidence implicating any one else. Such is the nature of the evidence against nerve-irritation in tetanus. The patients all die in the same way. Nerve-irritation is always present. Nerve-irritation has been convicted in at least three similar cases. The patients die in just the way nerve-irritation would kill them. The post-mortem appearances are just such as it would leave behind it. There is no evidence to show any other cause of death. In this simile I lose the value of the 5th proof, or that by treatment, which tells very strongly; but I think that the evidence is already strong enough.

HEREDITY AND CRIME IN EPILEPTIC CRIMINALS.

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I PURPOSE in the following tables to put together certain details relative to the hereditary history of those prisoners admitted into the Wakefield Gaol during the past three or four years, who were epileptics. The question is one of such interest in connection with the probable existence of a distinct criminal neurosis, that little apology, I think, is needed in bringing it to the attention of those specially interested in the study of mental disease. That there is some connection between crime and insanity few, doubtless, who have had any experience of either a prison or asylum would be prepared to deny, even if they were disinclined to go to the length of admitting that in many cases the two are distinctly correlated.

In making any inquiry into the history of prisoners there are three main difficulties to contend with. First: the well-known mendacity of the criminal classes generally. Secondly: the meagre and imperfect knowledge they usually possess of their relatives; and, Thirdly: the common occurrence of illegitimacy. In such an inquiry as the present the first difficulty is naturally one of the greatest moment.

It would occupy a needless amount of space to detail the precautions that have been taken in making these investigations, but it seemed right to put this great difficulty prominently forward, with the assurance that, so far as was practicable, every possible care has been taken to test the truth of the statements contained in the following pages.

Members of the class from which our criminals are mostly drawn commonly get separated from the parent stock very

early in life, and, in the subsequent struggle for existence, having to fight their way apart, rapidly lose all sight, and frequently all recollection, of even their immediate ancestors. In at least 5 per cent. of the cases examined the prisoners had no knowledge whatever of any member of their families. This difficulty applies especially to the Irish, who leave their country when comparatively young, and frequently never see or hear of any of their relatives again. As the Irish form about one-sixth of the whole number of prisoners admitted, it will be seen that this drawback is one of some moment. Perhaps the only compensating advantage is that the average results will be understated rather than exaggerated. The third difficulty is one that undoubtedly comes into frequent play, but is of such minor importance in comparison with the other two that it may be dismissed with the remark that inquiry has been made, and care taken to make such allowances for it as were necessary.

Out of a total of 119 cases examined, 89 were males and 30 females. It is usually taught that among the general population epilepsy attacks the two sexes in about equal proportion.[1] There is reason to think that with criminals the same rule holds good. During the year 1877 there were committed a total of 9672 males and 2832 females, or 1 female to every 3·3 males, a proportion, it will be seen, very similar to that between the two sexes among the epileptics.

The cases have been divided broadly into two classes—idiopathic and traumatic. This division is somewhat arbitrary; but the two classes exhibit a marked difference, both in hereditary and personal history. Any attempt to ascertain the exciting cause for epilepsy is generally a matter of difficulty even in intelligent patients; under the term traumatic, therefore, are classed only those cases in which the attack commenced for the first time within a month at least of some sudden psychical or physical accident. Under the head of idiopathic are placed all those where the fits had existed from birth, or commenced later on in life without the intervention of such rapidly acting external agents. As idiopathic cases, therefore, I have included all those assumed

[1] Reynolds's 'System of Medicine,' vol. ii. p. 295.

to be due to long-continued habits of intemperance on the part of the patients themselves; and this the more readily because certain forms of alcoholism with resultant epilepsy are undoubtedly due to such hereditary predisposing causes as, under other circumstances, or in individuals differently constituted, would have directly produced the convulsive attacks. Dr. Savage has seen cases that convinced him that drink-craving has been the result of nervous disease in one or other parent.[1] This division applies only to males. The ascribed traumatic causes among females were so few and slight that, for the sake of simplicity, they have all been counted as idiopathic.

Among the traumatic cases, 26 in number, 15 were said to be due to injury to the head, 5 to fright, 4 to sunstroke, and 2 to injuries of an undefined nature. These two last occurred early in life. In neither was there any distinct history of head-injury, but, in both, the fits followed immediately after the accident.

The ages of the epileptic prisoners are given in Table I., and in Table II. the ages of the 3341 prisoners of all classes committed during the quarter ending March 31st, 1878.

TABLE I.

Table showing the Ages of 119 Epileptic Prisoners.

Ages.	Idiopathic.						Traumatic.		Net Total.	
	Males.		Females.		Total.		Males.			
	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
16 and under 21	4	6·4	1	3·3	5	5·3	2	7·7	7	5·8
21 „ „ 30	13	20·6	10	33·4	23	24·8	6	23·1	29	24·4
30 „ „ 40	26	41·3	11	36·7	37	39·8	6	23·1	43	36·2
40 „ „ 50	11	17·4	4	13·3	15	16·1	8	30·7	23	19·3
50 „ „ 60	5	7·9	4	13·3	9	9·7	4	15·4	13	10·9
60 and upwards	4	6·4	..	..	4	4·3	..	..	4	3·4
Total . .	63	100·0	30	100·0	93	100·0	26	100·0	119	100·0

[1] 'Guy's Hospital Reports,' 1877, p. 68.

TABLE II.

TABLE showing the AGES of 3341 PRISONERS committed during the QUARTER ending MARCH 31st, 1878.

AGE.	Males.		Females.		Total.	
	No.	Per cent.	No.	Per cent.	No.	Per cent.
Under 12 years of age . .	6	0·2	1	0·1	7	0·2
12 and under 16	41	1·5	18	2·7	59	1·8
16 „ „ 21	301	11·2	110	16·5	411	12·3
21 „ „ 30	974	36·4	204	30·7	1178	35·3
30 „ „ 40	674	15·2	175	26·3	849	25·4
40 „ „ 50	361	13·5	115	17·4	476	14·2
50 „ „ 60	221	8·3	35	5·3	256	7·7
60 and upwards	98	3·7	7	1·0	105	3·1

A comparison of these two tables will show that the decennial period between 21 and 30 includes the larger number of ordinary prisoners, while that between 30 and 40 takes the greatest percentage of epileptics. The average age for epileptic males is 37·0; and for females 34·8. This greater average age for epileptic prisoners may be partly due, as will be seen further on, to their more prolonged and persistent criminal career, as indicated by the greater number of recidivists among them. The large percentage in the decennial period between 40 and 50 among the traumatic males is probably accidental, the number of cases being too small to furnish any decisive information.

Table III. shows the ages at which the fits commenced. Table IV. is taken, for the purposes of comparison, from Dr. Bennett's Clinical Lectures, and Dr. Reynolds's article in 'The System of Medicine.'[1]

[1] Vol. ii. p. 296.

TABLE III.

TABLE showing the AGES at which the FITS commenced in CRIMINAL EPILEPTICS.

AGE.	IDIOPATHIC.						TRAUMATIC.	
	Males.		Females.		Total.		Males.	
	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
Under 10 years . .	20	31·7	9	30·0	29	31·2	2	7·7
From 11 to 20 . .	18	28·5	5	16·7	23	24·7	8	30·8
„ 21 „ 30 . .	14	22·2	8	26·6	22	23·6	7	26·9
„ 31 „ 40 . .	8	12·7	3	10·0	11	11·8	4	15·4
„ 41 „ 50 . .	2	3·1	5	16·7	7	7·5	4	15·4
Over 50	1	1·6	..	..	1	1·1	1	3·8

TABLE IV.

TABLE showing the AGES at which the FITS commenced in Ordinary EPILEPTICS.

DR. BENNETT'S CASES.				DR. REYNOLDS'S CASES.			
AGE.	Males.	Females.	Total.	AGE.	Males.	Females.	Total.
From 1 to 10 .	9	14	23	Under 10 . .	10	9	19
„ 10 „ 20 .	11	23	34	From 10 to 20.	66	40	106
„ 20 „ 30 .	14	9	23	„ 20 „ 45.	25	20	45
„ 30 „ 40 .	10	6	16	Over 45 . . .	1	1	2
„ 40 „ 50 .	1	..	1				
„ 50 „ 60 .	2	1	3				
Totals . .	47	53	100	Totals . .	102	70	172

From the latter table it appears that in ordinary epilepsy the majority of cases commence between the tenth and twen-

tieth years of life, the numbers in the decennial period on either side of this being about equal. Trousseau also gives the first place to the second ten years of life, but states that a larger number of cases occur in the period before than in the period after.

The particulars in the column for traumatic cases in Table III. are of little interest. Being directly due to outside causes, the fits, as one would naturally expect, occur for the first time during those years of life when the body is most exposed to external injury. Those under ten years of age were the two boys already mentioned, and having a doubtful traumatic origin. Among the idiopathic cases the most noticeable point is the larger percentage commencing under ten years of age, which, in my own cases at all events, practically means that the fits had existed from childhood. M. Lancereaux found with regard to fits as the result of hereditary alcoholism that, in children who had survived the dangers of convulsions in infancy, epilepsy not unfrequently broke out at the period of puberty.[1] Three or four of my own cases go in support of this statement, an interval of several years elapsing between the cessation of the convulsions and the development of epilepsy proper.

In suggesting any explanation of this earlier age for the commencement of epilepsy among criminals, it should be borne in mind that the disease occurring early in life must be a great hindrance to the sufferers being brought up to any useful trade or mechanical employment, or to their competing with others who are not so afflicted; so that with the ordinary means of subsistence lessened, frequently with a tendency to weak-mindedness, and with no relatives capable of supporting them, it is little to be wondered at that they should gradually gravitate into a state of vagrancy and beggary. In a mining district like the West Riding this probably holds good to a greater extent than it would in an agricultural county. Many of these criminal epileptics are well-known characters, and spend the greater part of their life fluctuating between the workhouse and the prison.

Case No. 4. Male, æt. 33. Has had fits since he was 14 years

[1] 'Gazette des Hôpitaux,' April 26, p. 377.

old. They are of such frequency and severity that he has had to spend the greater part of his subsequent life in the workhouse, as no one would keep him employed for any length of time. He bites his tongue severely, and his face and head are profusely scarred from injuries he has received. They always commence with a prickling sensation in the right hand. Whenever he thinks a fit is coming on, he gets drunk, if possible, with the view of warding it off, but he does not think it has the desired effect. Father died suddenly thirteen years ago; was not a heavy drinker. Mother is living; neither drinks nor has fits. Brother was a heavy drinker. Youngest sister had a running from the ears and was "troubled in her head"—probably imbecile. Eldest sister had eleven children, all of whom died when three or four days old, mostly in convulsions. He himself is a drunkard, and has been committed to prison four times as a disorderly pauper, twice for larceny and twice for assault.

Case No. 90. Male, æt. 47. Had convulsions in childhood, which were said to be due to "water on the head." He has since been "affected in his nerves throughout his body." He gets his living now by singing hymns about the street. Before that he was kept at home, never having been taught any trade. About nine or ten years ago he had a stroke and was paralysed "all over his body," so that he was unable to hold a pen or do anything for himself. Prisoner is of weak intellect and has right hemiplegia. He has no settled residence and remembers little or nothing about his family. He has not seen any of them now for many years; but has heard that his father was a moderate man and died of old age, and that his mother died when he was a child. He is committed to this prison as a disorderly pauper. Has been in other prisons, but does not remember how many times.

It will be seen, on referring to Table XI., that the convictions under the heads of rogues and vagabonds, vagrancy and disorderly paupers are more numerous among epileptics than non-epileptics. Again, it will be shown in the next paragraph that a very frequent probable predisposing cause of epilepsy among criminals is drunkenness on the part of the parents. It is now a well-established fact that the children of drunkards are

especially liable to convulsions in infancy. M. Martin has lately investigated this question at the Salpétrière. In his first group of 60 epileptic patients, one-fifth of the children had convulsions, and more than one-half died early. His "83 families, in which one or more members suffered from epilepsy of alcoholic orgin, had 410 children; and of this number, 108 (more than one-fourth) have had convulsions; and in 1874, 169 were dead, 241 lived, but 83 (more than a third of the survivors) were epileptics."[1] It has been remarked by Dr. Reynolds that "where there is a marked hereditary taint as a predisposing cause of epilepsy, the disease is found to develop itself somewhat earlier than under other circumstances."[2] The difference for ordinary epileptics is represented in the following table:—

Commencing under æt, 15 . . .	83·33 hereditary	46·15	non-hereditary.
„ above æt. 15 . . .	16·66 „	53·82	„

The same rule is found to hold good with respect to criminals, but the difference is somewhat less marked. The distinction with regard to sex, on the other hand, is rather in accordance with the results arrived at by Messrs. Leech and Fox; hereditary epilepsy in criminals shows itself on an average four years earlier among men than among women.

Unfortunately, one of the most prominent features in the hereditary history of these prisoners is one concerning which there is the greatest difficulty in arriving at the truth with any degree of exactitude. The amount of intemperance thought sufficient to constitute a "drunkard" is found to vary very materially in the minds of different individuals. The "heavy drinker" of one son, is the father who "only gets drunk every Saturday night" of another, a practice thought to be so little out of the common, that the man is called "sober." As this seemed to be one of the most interesting points in connection with the subject, precautions have been taken to include under the head of "drunkards" none but those in whom the habit was marked and confirmed. Two classes of people have been specially noted, namely those described as "scarcely ever sober," and those subject to paroxysmal attacks

[1] 'Brain,' July 1879, p. 293. [2] *Op. cit.* p. 296.

of an uncontrollable character, with intervals of sobriety or even total abstinence; cases, in short, where the habit has become distinctly morbid. In ordinary phraseology, nearly all those marked as doubtful would come under the head of "drunkards." The difficulty in estimating the amount of drunkenness in individuals is so great, that some authors think it right to reject such evidence altogether. By so doing they appear to me to exclude one of the most valuable points in the inquiry. As it is more conceivable that patients should conceal drunken habits in other members of their family, rather than exaggerate or invent them, the tables will have at least the negative merit of understating the probable truth. As a matter of fact, in most cases the father was found to be the great drunkard of the family. A history of drink on the part of the mother was but rarely met with. This is probably simply an indication of the greater sobriety of the female half of the population, though the greater reticence of the prisoners with regard to their mothers' failings may have something to do with it.

TABLE V.

HABITS of the FATHERS of EPILEPTIC CRIMINALS.

	IDIOPATHIC.						TRAUMATIC		Net Total.	
	Males.		Females.		Total.		Males.			
	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
Fathers drunkards	35	55·6	11	36·7	46	49·5	7	26·9	53	44·6
Fathers sober . .	21	33·3	9	30·0	30	32·3	11	42·3	41	34·4
Unascertained or doubtful	7	11·1	10	33·3	17	18·2	8	30·8	25	21·0
Total . .	63	100·0	30	100·0	93	100·0	26	100·0	119	100·0

In addition to the details given in the above table, 15 idiopathic males—23·8 per cent.—gave a history of drunkenness in 24 other members of their family, distributed thus:—Grandfather, 1; mothers, 3; uncle, 1; aunt, 1; brothers, 18.

Three idiopathic females—10 per cent.—gave a history of drink in three other members of their family; in one case a mother, and in the other two brothers. One traumatic male said his mother had been accustomed to drink heavily, and had committed suicide while under its influence. One of the females' fathers also committed suicide.

The difference between the idiopathic cases and the traumatic is most marked; the former giving a total of 49·5 per cent. of fathers as drunkards, the latter only 26·9 per cent. One other point worthy of note in this table is that the percentage of drunken fathers among the males is greatly in excess of that among the females. Probably many of the doubtful cases among the latter should by right be included; but, making allowance for this and for the possible greater amount of drunkenness among the mothers, an hereditary history of alcoholism is certainly more frequent among epileptic men than women. This may be partly compensated for by the increased number of epileptics among the ancestors of the females. (See Table X.)

It is difficult to estimate the importance of this table without having some notion of the frequency of drunkenness in the parents of criminals who are non-epileptic. The only figures I have, selected with the same stringency, are those of 30 females, taken consecutively for this purpose; they give the following results:—

Fathers drunkards . . .	7 = 23·3	per cent.
Fathers sober	15 = 50·0	,,
Unascertained, or doubtful .	8 = 26·7	,,
Mothers drunkards . . .	2 = 6·6	,,
Mothers sober	23 = 76·7	,,
Unascertained, or doubtful .	5 = 16·7	,,

Among these women, then, with a less number of doubtful cases, there is a decrease of 13·4 per cent. of drunken fathers, and an increase of 20 per cent. of those who were said to be sober. A comparison of the two columns, idiopathic and traumatic in Table V., shows among males a still greater difference in the percentage of drunken fathers.

My colleague, the Rev. R. Bullock, Chaplain to the Prison, has kindly compiled for me a table of the presumed habits of

the fathers of 225 male prisoners committed for 6 months and over. This table gives:—

Fathers drunkards . . .	97 = 43·5	per cent.
Fathers sober.	126 = 56·5	„

The comparison perhaps is hardly just, as men committed for this length of time belong, as a rule, to a different class from those committed for short periods; but the difference is 12·1 per cent. in favour of the non-epileptics, a difference, it will be observed, remarkably similar to that found to exist between epileptic and non-epileptic criminal females. If equal stringency had been exercised in apportioning the habits of the fathers in these cases, the difference would in all probability be found to have been still greater on the side of the men.

Mr. Bullock has also furnished me with the statistics of all the boys committed to reformatories during the years 1872 to 1876 inclusive. The total number of boys was 730; those said to have had drunken fathers being 194, and drunken mothers 29. This will give a percentage of 26·5 for the former, and 3·9 for the latter. The percentage of drunkards among the mothers is, it will be observed, only slightly lower than it was shown to be among the idiopathic epileptics. I was prepared to find a considerably greater difference in the number of drunken ancestors between epileptic and non-epileptic criminals than was really proved to exist. There is reason, however, to believe that a hereditary history of alcoholism is more common, not only in epileptic but in ordinary prisoners than it is in non-criminal epileptics. Of 95 ordinary epileptics examined by M. A. Voisin, 12 had scrofulous or true tuberculous antecedents, and 12 had antecedents who died from alcoholic excesses, or were subject during their honeymoon to excessive abuse of alcohol.[1] This puts the proportion of drunken parents at only 12·6 per cent. Reasoning from the above facts, it is difficult to avoid the conclusion that both crime and epilepsy may owe their origin to alcoholism in the parents as a predisposing cause; in other words, that criminal instincts and epileptic convulsions are not unfrequently distinctly correlated. Nor do I see any reason to confine the term "criminal

[1] 'London Medical Record,' January 15th, 1878, p. 9.

instincts" in these cases to a tendency to alcoholic excess. Among the idiopathic males, there are 26 cases where the habits of the fathers were ascertained without there being any epilepsy or mental disease in the family. The average number of convictions for those with drunken fathers, as compared with those whose fathers were sober, is as 3·5 to 2·3, showing an increased amount of crime associated with hereditary alcoholism.

The statement that epilepsy in children is due to conception occurring while the parents are under the influence of drink, is one that must be naturally difficult of proof. I failed to get any evidence of the fact, nor do the conditions at all seem to require it. It is not suggested that the hereditary transmission of epilepsy is due to conception taking place during a paroxysm; the hereditary transmission of alcoholism then, which in some of its forms is surely an allied neurosis, would be guided by the same laws and occur under like conditions.

I pass on now to consider other forms of hereditary neurosis, the most important of which is naturally epilepsy itself.

Direct heredity in the case of criminals can seldom be traced beyond the father or mother. No general value therefore is to be attached to the few particulars given of grandparents.

Collateral heredity is confined mostly to brothers, sisters, uncles, aunts, and first cousins.

The idiopathic epileptic males give, including both direct and collateral relatives, a family history of fits in 29 cases, or 46 per cent. This includes one case in which the prisoner's children only were affected. The females give a like history in 21 cases, or 70 per cent., including two in which children only were affected; and the traumatic males in 4 cases, or 15·3 per cent. The total number of idiopathic cases giving a history of fits in one or more members of their family is then 50, or 53·7 per cent.; and the net total for all cases 54, or 45·3 per cent. Counting only direct relatives, we get a percentage of 25·8, 32·4, and 12·5 in the three classes respectively. The details are given in Table VI.

TABLE VI.

Details of Hereditary History of Fits (omitting Children).

IDIOPATHIC CASES.—Males.

Relative.	No.	Per cent.	Degree of Relationship when ascertained.	Remarks.
Grandfather	1	1·6	Mother's father.	
Grandmother	1	1·6	Father's mother.	
Fathers	4	6·4		
Mothers	10	16·2	.. ..	1 "died of fits."
Brothers	14	22·6	.. ..	8 "
Sisters	13	20·9	.. ..	5 "
Uncles	4	6·4	3 were Mother's brothers.	1 "
Aunt	1	1·6	Mother's sister.	
Cousins	3	4·9	1 on Mother's side. 1 Father's brother's daughter.	
Nephews and Nieces .	11	17·8	Sister's children.	All died in Infancy.
Total No. of Relatives with Fits	62	100·0		

Females.

Relative.	No.	Per cent.	Degree of Relationship when ascertained.	Remarks.
Fathers	4	11·7	.. ..	1 "died of fits."
Mothers	7	20·7	.. ..	1 " "
Brothers	7	20·7	.. ..	2 " "
Sisters	6	17·7	.. ..	1 " "
Uncles	4	11·7	2 were Mother's brothers. 1 was Father's brother.	
Aunts	4	11·7	2 were Mother's sisters.	
Cousin	1	2·9	Female.	
Nephew	1	2·9	Brother's child.	"Died of fits."
Total No. of Relatives with Fits . . .	34	100·0		

TRAUMATIC CASES.

MALES.

Relative.	No.	Per cent.	Degree of Relationship when ascertained.	Remarks.
Grandfather	1	12·5	Mother's father.	
Brothers	4	50·0	.. ..	2 "died of fits."
Sister	1	12·5		
Total No. of Relatives with Fits . . .	8	100·0		

PROPORTION of SEXES among RELATIVES AFFECTED.

IDIOPATHIC.				TRAUMATIC.	
Males.		Females.		Males.	
	Per cent.		Per cent.		Per cent.
Males . . .	23 = 37·1	Males . .	16 = 47·1	Males . .	5 = 83·3
Females . .	26 = 42·0	Females . .	18 = 52·9	Females .	1 = 16·7
Unknown . .	13 = 20·9				

Of the 29 cases among the idiopathic males in which epilepsy was present in the family, it existed in the father in 1; mother in 3; grandfather in 1; grandmother, mother, sister, brother, uncle, and child in 1; father and sister in 1; father and two sisters in 1; mother and sister in 1; mother and brother in 2; mother and five or six brothers and sisters in 1; mother, sister, and brother in 1; mother and aunt in 1; brother in 4; sister in 2; brother and cousin in 1; brother, uncle and two great-uncles in 1; sister and uncle in 1; uncle in 1; cousin in 2; ten or eleven nephews and nieces in 1; four children in 1.

Among the 21 idiopathic females, epilepsy existed in the father in 1; mother in 4; father, sister, cousin and child in 1; father, brother and child in 1; father and sister in 1; mother and child in 1; mother, brother and uncle in 1; mother and

brother in 1; brother in 1; brother, sister and child in 1; brother and aunt in 1; brother, sister, nephew and two aunts in 1; two sisters and uncle in 1; aunt in 1; uncle and six children in 1; two children in 1; three children in 1.

Among the 4 traumatic males, epilepsy existed in the grandfather, brother, and two children in 1; brother in 1; two brothers in 1; sister in 1.

With respect to numbers only, the 124 relatives were divided among the 54 epileptics as follows:—

Idiopathic Males.

1	had	11	relatives	affected.
2	„	5	„	„
1	„	4	„	„
2	„	3	„	„
8	„	2	„	„
15	„	1	relative	affected.

Idiopathic Females.

1	had	4	relatives	affected.
3	„	3	„	„
5	„	2	„	„
11	„	1	relative	affected.

Traumatic Males.

2	had	2	relatives	affected.
2	„	1	relative	affected.

An examination of the above tables, having regard to the idiopathic cases only, suggests the following conclusions: First; both among males and females epilepsy is more frequent in the mother than in the father: and secondly; the percentage for both parents is higher with the women than it is with the men. On comparing the above figures with those in Table V., it will be seen that this result is exactly the converse of what holds good with respect to drunkenness. There the male parent was found to be affected to a much greater extent than the female, and the percentage for both parents was higher among men than women. According to Trousseau, in ordinary cases hereditary predisposition is particularly apparent in persons descended from epileptic mothers.[1] The percentage of epileptics among brothers and sisters is slightly higher for

[1] Trousseau, Syd. Soc. Translation, 1879. Vol. iv. p. 357.

men than women, notwithstanding the fact that the number of direct hereditary epileptics is much greater among the latter. This seems to favour the suggestion previously made that certain forms of alcoholism in the parents are almost as efficient a predisposing cause for convulsions in the children as epilepsy itself; the number of drunken parents being so much greater among males than females. In connection with this point, and also as showing the great tendency of alcoholism to produce early convulsions in the offspring, it may be mentioned that the average age for the commencement of the fits, for those epileptics who have a direct hereditary history of drink, is less by 4½ years than for those whose parents are returned as sober.

Epilepsy in the father was usually found in connection with drunken habits. Out of 9 cases, where all the particulars were ascertained, epilepsy in the mother was associated with drunkenness in the father in 6, and the result was invariably intemperance on the part of the prisoner himself.

The large proportion of epileptics among the brothers and sisters in the traumatic cases would show that, apart from the exciting cause, there is often a marked hereditary predisposition. M. Lancereaux holds, with regard to alcoholic epilepsy, that the hereditary tendency of drunkenness manifests itself in the offspring in the form of excessive reflex excitability. He cites the case of a young boy who had severe epileptiform fits, lasting, with intermissions, for several hours, as the result of intestinal worms, the unusual severity of the attack being due to parental alcoholism.[1]

Case No. 3. Male, æt. 24. Was healthy until two years ago, when he got his head crushed by some machinery. Was removed to the County Infirmary, and five days afterwards he had his first fit. The fits have since occurred on an average about once a fortnight. They come on both day and night, and he has cut his face and bitten his lips several times. About two months ago he woke up one morning and found himself paralysed on the right side. His father was a heavy drinker. Two brothers died, æt. 7 and 18, both of whom had fits. Has an uncle who was in an asylum, and a cousin who is

[1] 'Gazette des Hôpitaux,' April 26.

there now. Acknowledges to having been a heavy drinker himself. Is in prison for the first time for larceny.

Case No. 17. Male, æt. 19. Twelve months ago he fell 7 feet on to his head, and was insensible for some time afterwards. Two days after the fall he had his first fit. They come on now at intervals of three or four weeks, and last but a few minutes. He bites his tongue freely, and has fallen down and cut his head more than once. Father is a heavy drinker. Mother living; does not have fits. One brother, aged 30, has had fits all his life. Prisoner himself is a heavy drinker. Has been committed to prison for damage, larceny, and drunkenness.

Case No. 18. Male, æt. 58. Was in the Indian Mutiny in 1858, and received a wound on the head; five or six years ago he fell and cut his head again in the same place. While the surgeon was examining this wound he had his first fit. The wound was followed by erysipelas. The fits have occurred since at very variable intervals, sometimes two or three a day, at others only one in four or five weeks. They last but a minute or so, and come on mostly in the night time. He bites his tongue and lips freely, and has cut his head severely. At the time of, or shortly after the fit, he is very violent, striking and biting any one near him. In consequence of these attacks of excitement he has been confined in an asylum on four different occasions, Father was lost at sea; was not a heavy drinker. Mother was a drunkard and drowned herself. She used to have fits, which, from prisoner's description, were probably attacks of *petit mal*. Has three brothers and one sister, the latter is subject to fits. His maternal grandmother was in an asylum. Is committed to prison for drunkenness. Has been in twice before, once for disorderly behaviour and once for assault.

Case No. 19. Male, æt. 45. When 19 years of age he had a fall from the rigging of a vessel; broke four ribs and his collar-bone, and injured his back and head. Was in his first fit when taken out of the water. Had a rapid succession of fits for four weeks, and was scarcely in his senses for three months afterwards. The fits have gradually lessened in frequency since the accident, and they now only occur once or twice in the month. He never bites his tongue, but has scars all over his

face and hands from injuries received in falling. Has had his face paralysed for some days after a fit. There is partial paralysis existing on the left side now. Mother dead 18 years from rheumatism. Father living; is a heavy drinker, and has been so for many years. His maternal grandfather had fits. A younger brother also had fits, and died from "disease of the brain and falling sickness." Sister has been in prison. Prisoner himself had two children who died in convulsions. He is a heavy drinker. Has been in this prison five times; once for larceny, once for hawking without a certificate, and three times for drunkenness. Has also been in Leeds Gaol for drunkenness.

Dr. Bennett in his analysis of 100 cases gives a family history of epilepsy in 26 per cent.; the father and mother being most frequently affected, and the brothers and sisters next in about equal proportion. It seems probable, therefore, that both hereditary and acquired epilepsy is far more common among criminals than it is among the general population. Cause and effect alternate in successive generations. As the parental taint of epilepsy may develop itself as criminal instincts in the offspring, so there is every reason to suppose epilepsy in the children may have its hereditary predisposition in some forms of habitual crime on the part of the parent. "The hereditary predisposition of an epileptic," says Trousseau, "may be traced merely to strange, nervous phenomena, perfectly different from epilepsy itself, whilst similar disorders may alone be manifested by his posterity, direct or indirect."[1]

The very large amount of hereditary neurosis that is frequently met with in these cases is somewhat remarkable.

Case No. 20. Female, æt. 22. Had convulsions in childhood, from which she recovered. Since her pregnancy has been subject to fits. Was under observation in the hospital for some time, and there is no question as to their epileptic character. Father was a drunkard. Mother living; neither has fits nor drinks heavily. Prisoner's maternal grandmother was insane. One brother had fits, and died in one when aged 22. One sister has had fits since childhood. Two sisters have

[1] Trousseau, New Syd. Soc. Translation. Vol. i. p. 87.

been in prison for drunkenness. Has two aunts now living who have fits. A cousin—mother's sister's child—is now in Hull Asylum. One of brother's children died in convulsions at 5 years of age. Prisoner herself is an intemperate woman, and has been in prison 13 times, including 3 for drunkenness. From her own statement most of the other offences were committed under the influence of drink.

The proportion of sexes among the epileptic relatives is shown in the latter part of Table VI. The 13 returned as unknown, in the first column, include 11 sisters' children in one case, all of whom died in infancy from convulsions. It will be observed that under the head of idiopathic, the number of females affected is slightly higher for both sexes. This also is the converse of what holds good with regard to drunkenness.

As I am mainly concerned with the hereditary causes of epilepsy among these criminals themselves, any neurosis in their children has only been incidentally alluded to. The following cases, however, may serve, in passing, to show the manner in which they, in their turn, are not uncommonly found to be affected. That the children of epileptic criminals are more liable to convulsions and other nervous affections than the children of ordinary epileptics, naturally follows from the increased amount of heredity that is found to exist among the epileptics themselves.

Case No. 10. Female, æt. 40. Has had fits for 6 years. No cause suggested, except fretting from prolonged imprisonment. They come on at very variable intervals, and nearly always in the night time. Never bites her tongue, but has cut her head severely in falling. Father dead 21 years; was not a heavy drinker. Neither of her parents, nor any other member of her family had fits. Prisoner herself is a drunkard. Has been in prison 14 times, including 7 for drunkenness, and two sentences of penal servitude of 5 and 7 years respectively. Her eldest daughter is a bad character, and has been in prison several times. Three other children died young from convulsions.

Case No. 4. Female, æt. 46. Has had fits 5 or 6 years. Does not remember either her father or mother. Has been in

prison 18 times, including 12 for drunkenness. Has only two children, and both are epileptic.

Case No. 108. Female, æt. 34. Has had fits since childhood. They have gradually lessened in severity, and are now only attacks of *petit mal*. She was early attacked with hemiplegia, and is still partially paralysed on the left side. Her uncle and father's brother were subject to fits; the latter is now in an asylum. Has had 8 children, 6 of whom died in convulsions when young. Prisoner was tried at the Assizes for attempting to murder her child, and acquitted on the ground of insanity. She had acute melancholia, with delusions, and on several occasions attempted to commit suicide.

Case No. 31. Male, æt. 52. Has had fits since he was 10 years old. They only come on about three or four times a year; he has cut his head in falling, but does not bite his tongue. Father was a heavy drinker in his younger days. Mother has been dead 30 years from paralysis. Had 3 sisters and 1 brother, but has not heard or seen anything of them since he was a child. Has been in prison 5 times; 4 for drunkenness and once for assault. Had 5 children; 4 died in childhood from fits, and the fifth, a son æt. 25, is now in an asylum. (See Table VII.)

Of the 16 cases among the idiopathic males in which insanity was present in the family, it involved the mother's cousin and mother's brother's child in 1; the mother's mother, and mother's sister in 1. Of the 9 cases among the females, it existed in the father's brother and mother's sister in 1; in the mother's mother and mother's sister's child in 1. Among the 3 traumatic males, a first and second cousin were affected in 1, and an uncle and cousin in 1. All the rest had each but a single member of the family reported as insane.

Having found the proportion of epileptic relatives to be so much greater with criminal than with ordinary epileptics, it was only to be expected that inquiry into their hereditary history with regard to insanity should give us a similar result. Dr. Bennett's investigations show only 5 per cent. of ordinary epileptics with insane relatives, as against 24 per cent. of my own cases. The figures in the above table are too few to furnish any satisfactory general conclusions, but it may be

TABLE VII.

TABLE showing the details of INSANITY in the families of EPILEPTIC CRIMINALS.

RELATIVES INSANE.	IDIOPATHIC.				TRAUMATIC.	
	Males.		Females.		Males.	
	No.	Degree of Relationship.	No.	Degree of Relationship.	No.	Degree of Relationship.
Grandfather	1	Mother's father (committed suicide).				
Grandmother	1	Mother's mother.	1	Mother's mother.		
Fathers .	2	.. ..	1			
Mother . .	1	(Died insane and paralysed.)				
Uncles . .	4	2 were Father's brothers.	3	2 were Father's brothers. 1 Mother's brother.	2	1 was Mother's brother.
Aunts . .	3	2 were Mother's sisters.	3	2 were Mother's sisters.		
Brother . .	1					
Sister . .	1	(? Congenital imbecile.)				
Child . .	1	Son.				
Cousins. .	3	1 Mother's brother's child. 1 Mother's cousin.	3	1 Mother's sister's child. 1 Father's cousin.	3	(1 an imbecile.)
Total No. of Relatives Insane .	18		11		5	
Proportion of Sexes among Insane Relatives . .	Males . . 9 = 50·0 per cent. Females . 6 = 33·4 Unknown . 3 = 16·6		Males . . 4 = 36·4 per cent. Females . . 4 = 36·4 Unknown 3 = 27·2		Males . . 2 = 40·0 per cent. Unknown . 3 = 60 0	

remarked that, both with males and females, the uncle, aunt, and cousin are most frequently affected; that among the men the hereditary history of insanity is greater on their own side, while among the women it is equal for the two sexes.

There are a few examples of other forms of nervous disease in the family, which remain to be mentioned to complete this part of the family history. Five mothers among the men and one among the women were paralysed, and one died from disease of the brain. Two fathers died from apoplexy and one from paralysis. One maternal grandfather among the idiopathic males committed suicide while insane. Two cases of suicide as the result of drink have been mentioned in an earlier part of the paper. One woman was committed three times for attempted suicide, and made numerous other attempts while undergoing her imprisonment.

Table VIII. gives such details as I have been able to collect concerning crime on the part of other members of prisoners' families.

TABLE VIII.

TABLE showing the number of CRIMINAL RELATIVES of EPILEPTIC PRISONERS.

Relative.	IDIOPATHIC.			TRAUMATIC.	Net Total.	Remarks.
	Males.	Females.	Total.	Males.		
Father . . .	1	..	1	..	1	For drunkenness.
Mother . . .	1	..	1	..	1	„
Uncles . . .	3	..	3	..	3	2 were Father's brothers.
Brothers . .	4	3	7	1	8	2 for drunkenness.
Sisters . . .	..	3	3	1	4	2 „ „
Cousins . . .	2	..	2	..	2	Father's brothers' children.
Child	..	1	1	..	1	Female.
Total No. of Relatives convicted of Crime	11	7	18	2	20	

There are specially evident reasons for prisoners concealing any information they may possess on this subject. The knowledge of previous convictions so materially influences the result of any future criminal investigation, that they are naturally extremely reticent on the subject, both with regard to themselves and their relatives also. The difficulty is almost

as great in getting information from outside sources. There is, therefore, every reason for believing that the above table exhibits results considerably below the actual truth. The table of reformatory statistics before referred to gives on this point results greatly in excess of those for these older and epileptic criminals. Part of this may be due to children having a better knowledge of their relatives than adults who have been separated from home for many years; and part from their not appreciating the advisability of concealing the information.

TABLE IX.

TABLE showing the stated amount of CRIME amongst the RELATIVES of CHILDREN who have been committed to REFORMATORY SCHOOLS during the YEARS 1872 to 1876, inclusive.

Year.	No. of Boys.	Members of the Family who have been in Prison.					
		Father.	Per cent.	Mother.	Per cent.	Other Members.	Per cent.
1872	122	16	13·1	2	1·6	13	10·6
1873	154	15	9·7	4	2·5	23	14·9
1874	149	16	10·7	7	4·7	30	20·1
1875	150	19	12·6	3	2·0	43	28·6
1876	155	10	6·4	..	..	35	22·5

The only points noticeable in Table VIII. are that the collateral relatives are naturally the most numerous, and that drunkenness has been the offence in all the cases in which the nature of the crime has been ascertained.

Table X. (p. 514) shows briefly the general results obtained in the preceding pages with respect to the family history. The first part includes both direct and collateral relatives, the second part the former only.

From the first part of Table X. it will be seen that in the idiopathic cases drunkenness among the males and epilepsy among the females are respectively the forms of family taint most frequently met with, the very large proportion of epileptics among the latter making the net totals practically the same for both. There is also an increase both

TABLE X.

TABLE showing the GENERAL RESULTS of the INQUIRY into the FAMILY HISTORY of EPILEPTIC CRIMINALS.

	IDIOPATHIC.						TRAUMATIC.		Net Total.	
	Males.		Females.		Total.		Males.			
	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
With family history of drink	36	57·1	11	36·6	47	50·5	8	30·7	55	46·2
With family history of fits	29[1]	46·0	21[2]	70·0	50	53·7	4	15·3	54	45·3
With family history of insanity	16	25·4	9	30·0	25	26·8	3	11·5	28	23·5
With family history of crime	6	9·5	6	20·0	12	12·7	2	7·6	14	11·7
With direct hereditary history of fits or insanity	24	38·1	20	66·7	44	47·3	5	19·2	49	41·2
Without direct hereditary history of fits or insanity	39	61·9	10	33·3	49	52·7	21	80·8	70	58·8
With direct hereditary history of fits, insanity, drink, or crime	47	74·6	21	70·0	68	73·1	9	34·6	77	64·7
Without direct hereditary history of fits, insanity, drink, or crime	16	25·4	9	30·0	25	26·9	17	65·4	42	35·3

[1] One had children only affected. [2] Two had children only affected.

of crime and insanity among the relatives of the females. This great preponderance of hereditary mental disease among the women as a predisposing cause of epilepsy and crime is seen perhaps better in the latter half of the table. Counting only direct relatives, 66·7 per cent. had one or more members affected with fits or insanity, as against 38·1 per cent. of the males. This result, I believe, accords with the statement usu-

ally made in speaking of hereditary insanity, that the female sex has a specially marked tendency to receive the taint from the parents. Classing together all possible predisposing causes, namely drunkenness, epilepsy, crime, and insanity, there is a difference of only 4 per cent. between the sexes. There is every reason to assume, judging from the conclusions arrived at by writers on mental disease, that if we could carry the family history of these criminals beyond the first generation, this amount of hereditary predisposition would be enormously increased. The amount of hereditary neurosis in these cases as they stand, however, is greatly in excess of that usually stated to exist by most authors. Dr. Reynolds found only 12 per cent. of epileptics giving a distinct history of epilepsy in other members of their family, and in only one-third of the total number of cases was there any nervous disease in either their direct or collateral relatives.

Hughlings-Jackson has "very little faith in the hereditariness of such symptoms as epilepsy or convulsions."[1] He cites a case where fits were known to exist in four members of one family, and remarks "that instances so striking are rare." Among criminals even more marked cases than this would appear to be far from uncommon. Since the above tables were made out, a man has been committed for trial, and is now in the hospital as a confirmed epileptic. The following is his hereditary history: Father had fits all his life, and died in one when prisoner was a child. He had been a very heavy drinker. Mother died 12 years ago from heart disease; was not subject to fits, and did not drink heavily. Has 6 brothers living; one is a heavy drinker, and epileptic, the others all healthy. Has had 4 sisters; 2 died from fits, aged 11 and 37 respectively. The latter was married, and had 6 children, all healthy. The two sisters living are both married, one has 6 and the other 7 children. In the elder sister's family one child has fits, and another died young in convulsions. An uncle—mother's brother—was subject to fits, and died from "fits and dropsy." Two of his own children had fits when young, and both are now dead. One cousin—a girl, æt. 14, daughter of another uncle, also on the maternal side—was in

[1] Reynolds's Syst. Med. vol. ii. p. 271.

an asylum, and died there from "decline." Prisoner himself has had fits all his life, and has been a heavy drinker for many years.

The question as to the relative amount of hereditary epilepsy among the different classes of society is doubtless a difficult one to answer. Dr. Reynolds is inclined to think that "hereditary taint is more frequently discoverable among the better conditions of life than among the poorer." I cannot help thinking that at the extreme lower end, at all events, the amount of hereditary nervous disease is much greater than is usually supposed. It may be true that the lower classes have become more habituated to such active determining causes as anxiety, alarm, and want; but it is probable that this would be more than counterbalanced by the increased amount of physical injury to which they are subject, to their long-continued habits of intemperance, and especially to the marked influence of hereditary alcoholism.

Passing now from the family history of these epileptics, it is interesting to inquire if there is any difference between them and ordinary prisoners in the character and amount of their crime, as evidenced by the number and nature of their convictions. I shall endeavour to be as brief as possible, but the question is important as furnishing corroborative evidence of many of the conclusions arrived at in the preceding pages.

Table X. gives, in a few well-defined groups, the number and nature of the offences for which these epileptics were committed to prison. Table XI. shows, under the same headings, the total committals for the year ending September 30th, 1876. I have selected 1876 for comparison, as a larger number of epileptics came under my observation during that year than in any other.

It should be remembered in connection with Table X. that committals to Wakefield Prison are alone referred to. Many epileptics gave a history of previous convictions in other gaols, but—for the better comparison of these cases with ordinary criminals—all such convictions have been omitted.

TABLE XI.

TABLE showing the NUMBER and NATURE of OFFENCES committed by EPILEPTIC PRISONERS.

Offences.	IDIOPATHIC.						TRAUMATIC.		Net Total.	
	Males.		Females.		Total.		Males.			
	No. of Convictions.	Per cent.	No. of Convictions.	Per cent.	No. of Convictions.	Per cent.	No. of Convictions.	Per cent.	No. of Convictions.	Per cent.
Felony	45	17·7	28	10·5	73	14·0	14	22·6	87	14·9
Rogues and Vagabonds and Vagrancy	45	17·7	46	17·2	91	17·5	2	3·2	93	15·9
Neglect of Family	2	0·8	..	..	2	0·4	..	..	2	0·3
Disorderly Paupers	16	6·3	16	5·9	32	6·1	6	9·7	38	6·5
Common Prostitutes	..	..	9	3·4	9	1·7	..	..	9	1·7
Assaults	38	14·9	10	3·8	48	9·2	11	17·7	59	10·1
Wilful Damage	7	2·8	7	2·6	14	2·7	2	3·2	16	2·7
Drunkenness	80	31·5	124	46·5	204	39·2	20	32·3	224	38·4
Bastardy Laws	8	3·2	..	..	8	1·5	..	..	8	1·5
Other Summary Convictions	13	5·1	27	10·1	40	7·7	7	11·3	47	8·0
Totals	254	100·0	267	100·0	521	100·0	62	100·0	583	100·0

Under the head of "Other Summary Convictions," in Tables XI. and XII., the epileptics are included one for each among the following offences. Fraud; obtaining living by dishonest means; non-payment of rates; hawking without a certificate; embezzlement; field robbing; keeping unregistered lodging-house; poaching; ill-treating a pony; resisting a constable; warehouse breaking; unlawful pawning; attempt at murder; travelling without a ticket; two for neglect of family; three for attempted suicide; and six for breach of bye-laws.

On comparing Tables XI. and XII., it will be observed, so far as the idiopathic males are concerned, that the proportionate

TABLE XII.

Offences of Prisoners committed during the Year ending September 30th, 1876.

Offences.	Males.		Females.		Total.	
	No.	Per cent.	No.	Per cent.	No.	Per cent.
Felony	1401	16·3	437	16·0	1,838	16·2
Rogues and Vagabonds and Vagrancy	1141	13·3	213	7·9	1,354	11·9
Neglect of Family. .	167	1·9	3	0·1	170	1·5
Disorderly Paupers .	370	4·3	19	0·7	389	3·5
Common Prostitutes .	..	..	363	13·4	363	3·1
Assaults	1059	12·3	119	4·4	1,178	10·4
Wilful Damage . .	247	2·8	56	2·0	303	2·7
Drunkenness . . .	2683	31·3	1074	39·4	3,757	33·2
Bastardy Laws . .	99	1·1	..	..	99	0·9
Other Summary Convictions	1439	16·7	441	16·1	1880	16·6
Total	8606	100·0	2725	100·0	11,331	100·0

number of committals is greater among the epileptics for all offences except the following:—Neglect of family; wilful damage; and other summary convictions. For wilful damage the numbers are equal. An explanation for the increase among the vagrants and disorderly paupers has been already suggested. The percentage of committals for drunkenness is not appreciably greater. The year 1876 appears to have borne a higher percentage of committals than usual for this offence among the men. This is unfortunate, as there is reason to think that, as a rule, the proportionate percentage should be greater for epileptics than for non-epileptics. The last return I have by me, namely that for the quarter ending March 31st, 1878, gives the committals for drunkenness among the males at 24·6 per cent., or 7 per cent. below that of the epileptics. At the same time it must be remembered that drunkards are

often convicted of offences other than drunkenness, which have been committed simply under the influence of drink. The increased percentage under the heads of Felony and Assaults may owe its origin partly to this cause.

Turning our attention to the column for females, it will be observed that there is an increased percentage of committals among the epileptics for rogues and vagabonds, vagrancy, disorderly paupers, wilful damage, and drunkenness; and a decreased percentage for felony, common prostitutes, assaults, and other summary convictions. The increase in the first two classes of offences will probably be due to the cause suggested for the same fact in the male cases. With the increased percentage for drunkenness there is a decrease for assaults and felony. The total results for the idiopathic cases of both sexes show an increase for rogues and vagabonds, vagrancy, disorderly paupers, and drunkenness; and a decrease for felony, neglect of family, common prostitutes, and other summary convictions. The figures for wilful damage are the same. We might have expected to have found an increase for neglect of family; the reason for the decrease is, probably, that fewer of these prisoners are married, the existence of epilepsy not appearing to be a prepossessing feature. The number of committals among the traumatic males is too few to furnish any satisfactory conclusions when treated by themselves. The net totals for epileptics of all classes show an increase for rogues and vagabonds and vagrancy, disorderly paupers, drunkenness, and bastardy; and a decrease for felony, neglect of family, common prostitutes, assaults, and other summary convictions. The larger proportionate number of offences, then, for which these epileptics were committed appear to be those connected especially with drunkenness and vagrancy.

It has been asserted by Taquet that "sexual desires show themselves early in children of drunkards, and are associated with an absence of moral sense."[1] It is interesting, in connection with this statement, to observe that the percentage of convictions for bastardy is three times as great among the epileptics as it is among the non-epileptics. The interest of course lies in assuming that the epilepsy owes its origin to the

[1] Med. Record, Jan. 15, 1876. Page 8.

hereditary alcoholism which existed in all these prisoners. The above remarks, it must be remembered, apply only to the *comparative* number of convictions for the different offences. Inquiry into the *actual* number shows that not only are certain crimes more frequent among epileptics, but that the total number of convictions is considerably greater in all cases, both for males and females.

TABLE XIII.

TABLE showing AVERAGE NUMBER of CONVICTIONS for EPILEPTIC PRISONERS.

	IDIOPATHIC.			TRAUMATIC.	Net Total.
	Males.	Females.	Total.	Males.	
Number of Prisoners . . .	63	30	93	26	119
Total Number of Convictions	254	267	521	62	583
Average Number of Convictions	4·0	8·9	5·6	2·3	4·9

The difficulty in giving a similar table for ordinary offenders lies in the fact that in prison returns the number of *convictions* for any given time is stated, but not the number of *prisoners* convicted. One man, therefore, may be counted over three or four times, if he has been committed more than once during that period. I have specially examined the returns of about 2500 prisoners, discharged consecutively during the latter part of the year 1877, and eliminated this source of error so far as was practicable. The following are the results, it being premised that the average number of convictions given for each sex should, if anything, be lower than that stated:

	Males.	Females.
Total number of Prisoners	2072	501
„ „ „ Convictions	5957	2941
Average number of Convictions per Prisoner .	2·8	5·8

Prisoners convicted of drunkenness are, as a rule, sentenced

to short terms of imprisonment. The offence among chronic tipplers being frequently repeated, it follows that the number of previous committals for habitual drunkards would necessarily be in excess of that for other classes of prisoners. As there is little doubt about drunkenness being the more frequent offence among epileptics, it seemed advisable to discover, if possible, whether the increased average number of convictions for epileptics was due simply to the increased amount of drunkenness among them. It is impossible to determine the point accurately, on account of drunkards being so frequently committed for other offences. But I went carefully through the above returns for the females, as they are the chief offenders in this respect, with the following result:—Out of the 501 cases, 177 appeared to belong more especially to the drunken class. The total number of convictions for them was 1317, which would give an average per prisoner of 7·4, a considerable increase, it will be observed, on the numbers stated above, but still it does not reach that for the epileptics by about 1½. If the drunken epileptics only were selected, the difference would be increased by at least twice as much again. It is impossible, therefore, to avoid the conclusion that the amount of crime, as indicated by the number of convictions, is greater among epileptics than ordinary criminals. The difference in the present series of cases is really very marked. The number of convictions for the 501 females was 2941; supposing these women to have had each the same average number of convictions as the epileptics, this total would have to be increased by 1517.

The same conclusion may be arrived at by comparing the number of first committals and recommittals. Table XIV. gives the particulars for the epileptics, and Table XV. for prisoners of all classes. It must still be remembered, with respect to the latter table, that if a prisoner has been recommitted during the quarter, he will have been counted more than once.

TABLE XIV.

TABLE showing the number of FIRST COMMITTALS and RECOMMITTALS of EPILEPTIC PRISONERS.

Previous Committals.	IDIOPATHIC.						TRAUMATIC.			
	Males.		Females.		Total.		Males.		Net Total.	
	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
Once	12	29·3	5	23·8	17	27·4	5	41·7	22	29·7
Twice	6	14·6	..	..	6	9·7	3	25·1	9	12·2
Thrice	5	12·2	1	4·8	6	9·7	1	8·3	7	9·5
4 times	3	7·3	1	4·8	4	6·4	1	8·3	5	6·7
5 times	3	7·3	..	..	3	4·9	1	8·3	4	5·4
6 and 7 times . .	5	12·2	2	9·5	7	11·3	..	..	7	9·5
8, 9, and 10 times.	3	7·3	2	9·5	5	8·1	..	..	5	6·7
Above 10 times .	4	9·8	10	47·6	14	22·5	1	8·3	15	20·3
Total Recommittals	41	65·0	21	70·0	62	66·7	12	46·2	74	62·1
First Committals .	22	35·0	9	30·0	31	33·3	14	53·8	45	37·9

The number of recidivists among the epileptics is greater by 16 per cent. and 13·1 per cent. respectively for the idiopathic and traumatic males, and by 5 per cent. for the females; the totals being an increase of 14·5 per cent. for all idiopathic cases, and of 9·9 per cent. for epileptics generally. In Table XIII. both the idiopathic and the net totals, compared with Table XIV., show a smaller percentage of prisoners with from 1 to 5 previous convictions, and a larger percentage for all numbers over 5. The total number of prisoners with more than 10 previous convictions is greater by 5 per cent. for the epileptics, but the increase, it will be observed, is due entirely to the females, who show an excess of no less than 18 per cent.

TABLE XV.

FIRST COMMITTALS and RECOMMITTALS of PRISONERS received during the QUARTER ending MARCH 31st, 1878.

Previous Committals.	Males.		Females.		Total.	
	No.	Per cent.	No.	Per cent.	No.	Per cent.
Once	441	33·7	90	20·8	531	30·5
Twice	210	16·0	41	9·5	251	14·4
Thrice	154	11·8	39	9·0	193	11·1
4 times	115	8·8	42	9·7	157	9·0
5 times	73	5·6	24	5·6	97	5·6
6 and 7 times	104	7·9	46	10·6	150	8·6
8, 9, and 10 times . . .	71	5·4	21	4·9	92	5·3
Above 10 times	142	10·8	129	29·9	271	15·5
Total Recommittals . . .	1310	49·0	432	65·0	1742	52·2
First Committals	1366	51·0	233	35·0	1599	47·8

Of the 10 idiopathic females with more than 10 previous convictions:—

1 was committed 51 times, including 26 for drunkenness.
1 " 36 " 26 "
1 " 27 " 5 "
1 " 18 " 12 "
1 " 18 " 10 "
1 " 14 " 7 "
1 " 13 " 3 "
1 " 12 " 9 "
1 " 12 times, all for drunkenness.
1 " 17 times.

Of the 4 idiopathic males:—

1 was committed 30 times, including 21 for drunkenness.
1 " 22 " 7 "
1 " 12 " 3 "
1 " 11 times.

The 1 traumatic male:—

was committed 14 times, including 8 for drunkenness.

These 15 cases have an average of 24 convictions each, including 9·1 for drunkenness.

In Table XVI. are given the details of the habits of the prisoners and their fathers in the case of epileptic and non-epileptic males. The figures for the latter are part of the table before referred to as having reference to prisoners committed for 6 months and over.

TABLE XVI.

TABLE showing the HABITS of the PRISONERS and their FATHERS in the Case of EPILEPTIC and NON-EPILEPTIC MALES.

	Epileptic.	Non-Epileptic.
	per cent.	per cent.
Number of Prisoners of intemperate habits . . .	75·7	66·0
Fathers drunkards . . .	67·8	51·5
„ sober	32·2	48 5
Number of Prisoners of temperate habits. . . .	24·3	34·0
Fathers drunkards . . .	66·6	30·9
„ sober	33·4	69·1

I do not wish to lay too much stress on these figures, as the records of the epileptics are somewhat imperfect on this point, and also because the two classes of prisoners are not strictly of the same criminal type; but the table seems to lead to the following general conclusions:—

(1) There are more drunkards among the epileptics than among the non-epileptics, the proportion of temperate to intemperate prisoners among the latter being as 2 to 1, and among the former as 3 to 1.

(2) Among the non-epileptics, the percentage of drunken fathers is greater for prisoners of intemperate habits, and the percentage of sober fathers greater for prisoners of temperate habits.

(3) Among the epileptics, the percentage of drunken and sober fathers is practically the same for temperate and intemperate prisoners.

(4) The percentage of drunken fathers among the epileptics is greater, both for temperate and intemperate prisoners, than it is for the intemperate non-epileptics.

With regard to the epileptic females, the case is a little different; the number of drunkards among them is even still greater than among the men. It is the exception to meet with an epileptic female criminal who is not at the same time intemperate. Among these intemperate epileptic women the percentage of sober fathers is greater than the percentage of drunken fathers. It is probable that a more perfect acquaintance with their family history would show a larger proportion of drunkards among the mothers, but, making allowance for this, there is little doubt that the amount of hereditary alcoholism is considerably less with the women than it is with the men. On the other hand, we find that the amount of nervous and mental disease in the family is greater by at least 20 per cent. Whereas, then, epilepsy in men is associated especially with alcoholism in the parents, in women it is found more frequently in connection with epilepsy and insanity in other members of the family.

TABLE XVII.

TABLE showing the Number of EPILEPTIC PRISONERS suffering from other Forms of NERVOUS or MENTAL DISEASE.

DISEASE.	IDIOPATHIC.				TRAUMATIC.	
	Males.		Females.		Males.	
	No.	Per cent.	No.	Per cent.	No.	Per cent.
Confined in Asylum	3	4·7	4	13·3	2	7·6
Demented or Imbecile	2	3·0	..	..	1	3·8
Hemiplegie	8	12·7	1	3·3	2	7·6
With Facial Paralysis	2	3·1	..	..	..	3·8
Cripple from Birth	1	1·5	..	..	..	..
Deaf and Dumb	..	..	1	3·3	..	..
Wasting of limbs from Infantile Paralysis.	..	..	1	3·3	..	..

It is further noticeable, that the number of convictions for the intemperate epileptics of both sexes is greater than for the

temperate; the excess being due to the increased committals for drunkenness, and, as one would naturally expect from the foregoing, affecting the women to a much greater extent than the men.

In addition to their epileptic convulsions and frequent drunken habits, several of these prisoners were affected with other forms of nervous disease. (See Table XVII., p. 525.)

TABLE XVIII.

	Cases.											Summary.
	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	
Sex (M. Male, F. Female)	M.	M.	M.	M.	M.	M.	M.	M.	M.	M.	F.	10 M., 1 F.
Age	60	50	53	33	39	25	42	76	24	23	33	
Class (I. Idiopathic, T. Traumatic)	I.	I.	T.	I.	I.	I.	I.	I.	T.	I.	I.	9 I., 2 T.
Duration of Fits in years	24	22	15	33	8	25	3	76	2	15	4	
Duration of Hemiplegia in years	24	22	10	(?)	8	25	3	2	½	5	(?)	
Side affected (R. Right. L. Left)	L.	L.	R.	L.	L.	R.	L.	L.	R.	R.	L.	7 L., 4 R.
Habits of Prisoner (D. Drunkard, S. Sober)	D.	S.	S.	S.	..	..	D.	S.	D.	..	..	3 D., 4 S.
Habits of Father . . .	D.	D.	S.	D.	S.	(?)	D.	S.	D.	S.	S.	5. D., 5 S.
Habits of Mother. . .	..	..	D.	..	..	..	..	..	..	..	..	1 D.
Other members of family Drunkards	..	..	..	1	..	..	..	..	..	..	..	
Total No. of Convictions	30	3	1	2	5	1	1	8	1	1	1	
No. of Convictions for Drunkenness . . .	21	..	..	..	1	..	..	..	..	..	1	
No. of Relatives with Fits	1	..	..	6	2	..	..	2	2	..	..	
No. of Relatives Insane	..	..	..	1	2	..	1	1	2	..	..	

Of the 9 who had been insane, 3 men and 3 women had been confined in an asylum once, 1 man twice, 1 woman three times, and 1 man four times. In addition to the above cases of distinct mental disease, many of these epileptics are

more or less weak-minded, being dirty in their habits and idle at their work; they are occasionally given to emotional outbursts, and exhibit a general want of moral self-control. The existence of so large a number of cases of paralysis is somewhat remarkable. The ordinary temporary hemiplegia following an epileptic fit is not here referred to. All the 11 prisoners in the above table had organic hemiplegia at the time they came under observation. It is further remarkable that, in the large majority of cases, 7 out of 9 among those returned as idiopathic, the hemiplegia was on the left side. The principal details in connection with these cases may be gathered from the tabular analysis opposite (Table XVIII.).

Critical Digests and Notices of Books.

A Defence of Philosophical Doubt, being an Essay on the Foundations of Belief. By ARTHUR JAMES BALFOUR, M.A., M.P. London: Macmillan and Co., 1879. 8vo. pp. 355.

THE author announces in his Preface that his work "may be sufficiently described by saying that it is a piece of destructive criticism formed by a series of arguments of a highly abstract character,"—an awful announcement, as he seems to think, and as deterrent as if it had run, "Who enters here must leave Belief behind." When we do enter, however, into this cave of doubt, it is found to be not quite so dark as the threat implies—the possibility of belief up to a certain practical point being left as a kind of residuum of the destructive analysis; so that, notwithstanding that absolute certainty about anything is unattainable, a healthy man may yet eat his dinner in the reasonable expectation of digesting it. The philosophic question raised is whether beyond the limits of observation the universe is under the same reign of law as we observe it to be within them. It may, or it may not be—and this "may not" supplies the main force of this critical attack.

The only possible foundation of science being the belief in the law of universal causation, and this fundamental law itself being only provable from particulars by observation and experiment, it follows that if particulars which are beyond the range of observation and experiment may contradict the law, the universal truth of any scientific fact cannot be assumed to be absolutely free from a kind of doubt, which one may if one pleases call philosophical.

There may be no real doubt in the mind of any student of science that the law of causation is universal, but the possi-

bility of imagining doubt must, we think, be admitted to have been shown by our author. And this distinction leads us to ask what he means by philosophic belief and philosophic doubt? Something like an explanation is found at page 5, where the author draws what he considers a most important distinction between the causes which produce a belief (or doubt?) and the grounds which justify one. The causes, he says, are psychological, the grounds or reasons are philosophical. The author admits that "in constructing a philosophy a previous psychological enquiry may be required," but he denies that the causes of belief include the grounds for it, and that psychology can comprehend philosophy and all other mental actions. The science of mind may prepare the ground for a philosophy, but cannot, he says, add one fragment to its structure. That, however, must depend upon what is called the science of mind, or psychology. If it includes the knowledge of mental powers and aptitudes, the assertion is incorrect, and the error has an important bearing on the criticism on Mill, whose logic is adapted to mind as it exists with limitations of experience; whereas the demands of the author are founded upon mind as it may be conceived without limitations of experience and of knowledge. As a philosophical exercitation the criticism is successful, as showing that Mill's system does not and cannot prove the absolute and entire truth about Nature, that is, the Universe in all its conceivable extension in time and space; that it cannot even prove the universal existence of the law of causation or of uniform succession, much less that of any of the subsidiary laws dependent thereupon. But what then? It shows that man, not being omniscient, may perhaps conceive a philosophy founded upon the assumption that he is so, but which in his present state must remain a dream of the imagination, like the cap of wisdom and the sword of sharpness, and the three-league boots of Jack the Giant Killer.

In the meantime the author comforts us with the assurance that

"In all cases of Induction we can do no more than prove a certain law to be *probable*. If our observations or experiments be numerous and successful, the probability proved may be a very

high one; if they are few and ambiguous, it may be a very slight one; but in either case, what we prove is probability, and probability alone. This, however, need cause us no uneasiness. If demonstrative certainty is denied us, we may still by this method obtain that practical certainty which is all we require to guide us in the affairs of life."

This also seems the opinion of Professor Jevons, and, after all, the *laches* of which Mr. Mill appears to be convicted are, that by avoiding to state the philosophical shortcomings of the method of proof from particulars, he has so stated his position that it cannot in reality "for a moment stand against hostile criticism." Considering what Mill has done to improve the method of ratiocination which enables us to "attain all that practical certainty which is all we require to guide us in the affairs of life," the judgment does seem heavy for the offence.

The author supposes himself to be asked whether the existence of a persistent universe, governed by causation, really does require proof, seeing that the belief in such an universe lies at the root of all knowledge, and the proof of it is impossible and unnecessary; and he replies that, while as a matter of fact, the law of causation does not appear to be accepted in its integrity by the greater part of the human race, and that those who do accept it seem to feel the necessity of founding it upon some kind of proof; with regard to the persistent universe, practically every one does believe in it, even those who speculatively question it. But is it possible to believe in a persistent universe without that belief which seems necessarily complementary to it, in the constant succession of events, which is the law of causation, and the foundation of all law? Persistent universe without law seems inconceivable.

The objection, from the occurrence of optical or other so-called illusions of the senses, which the author takes to the immediate belief which he admits that the majority of mankind certainly have in the testimony of their perceptions, seems to us forced and untenable. Granted that this evidence is sometimes untrustworthy, and that perceptions may arise in us which have no correspondent objects, it is of the very essence of the method of induction to eliminate such sources of error; and an inductive philosophy of perceptions will

not confuse those which are false and illusory with those which will bear the tests of verification. It is curious that after taking this objection to a knowledge of the universe, founded upon illusions of the senses, the author should be able so clearly to state the great difference between thought which has a true relation to sense, and thought which has not. "Grant that everything which is real is thought, it cannot be the fact that everything which is thought is real, since if it were so, mistakes as to the true nature of any object would be impossible; a doctrine as subversive of science as any form of idealism ever devised."

The obvious answer to this paradox is that everything which is thought is real as thought, and that the examination of all thought, even of that which has no known correspondence with outside causes of thought, is a proper and by no means subversive subject of mental science.

The middle chapters are devoted to the Transcendentalism of Kant and the Authority of Consciousness of Hamilton; but it is hard to understand the necessity for discussing these metaphysics in relation to scientific beliefs. The following three chapters, in which Mr. Herbert Spencer is belaboured, keep closer to the professed object of the work, although they come more near than need be to a general and unfavourable criticism of Mr. Herbert Spencer's opinions; the conclusion of which is that although Mr. Spencer has an astonishing faculty for collecting from every department of knowledge the facts which tell in his favour, his sensibility is blunted in the matter of difficulties and contradictions, and on metaphysical ground he is a trespasser. It cannot, however, be denied that in the Inconceivability of the negative Mr. Spencer has supplied just that axiomatic test of certitude which is demanded by philosophical doubters or debaters, a test whose main value may be that of an intellectual gag, but as such firm and efficient. At the twelfth chapter we somewhat escape from the torrent of destructive criticism, and are allowed to set foot on the solidifying ground of the author's own opinions. Considering science as a logical system, he tells us that "among bodies scattered through space is assigned to each man one of especial importance to himself, his own organism." "The

peculiarity about them [these organisms] is that they are the immediate channels of connection between the world I have just described and the thinking beings who by their means are made acquainted directly with the appearance of that world, and indirectly with its nature and constitution," p. 243. This description by one who shares the "implicit and indestructible confidence in the truth of that which had to be proved" by science, is not a little remarkable, and leads to surmise as to what the author's psychology may be, which induces conviction in the overwhelming scientific evidence of the truth that "all beliefs are the result of the operation of natural causes, and of these alone," p. 274, and in that "physiology which shows it (the evolution of belief), *à priori*, by demonstrating the dependence of thought upon the organism, and of the organism in inheritance and environment," p. 261. It was possible that the author might maintain that all beliefs are the result of natural laws acting upon the organic channels of communication which exist between the world, that is the environment, and of the thinking beings which are at the other end of the channels; but by ordinary persons the evolution of belief by natural and physiological processes would seem to be inconsistent with power of regarding each man's own organism merely as a channel of communication between the world and the thinking being, that is to say, between the environment and his real self.

It must be over subtle or merely fanciful to say that scientific notions are anthropomorphic in the same sense that our ideas of the Deity are so, and if the former the point is too fine to be felt. The idea of inorganic force has sometimes been very clumsily likened to that of animal effort, but it would be difficult to say what other scientific notions can by any straining of imagination be called anthropomorphic. Yet this is the leading argument in one of the author's most intelligible chapters—that on Science as a Logical System. Another objection to belief in our perceptions is that, while some of our observations of the qualities of bodies are apparently true, as that of extension, others, as that of the quality of colour, are not true; bodies being themselves devoid of colour, which resides in the vibrating medium and

the organs. Great capital is made out of this, which is merely a bit of slipshod wordsigning. If it be not said that a violet is blue, but that a violet is bluecausing, the statement would be as scientifically accurate as that a violet is soft and small, and all the philosophic fog raised by the author about the untrustworthiness of scientific observation about coloured bodies would disperse. Moreover Science, and Science alone, does provide us with means of distinguishing perceptions which are immediate from those which are mediate or altered, and those which have an object from those which are illusory; to distinguish the author's moonshine, for instance, to which he makes a special reference at page 255, from the brain-begotten vision of a ghost, which he calls *optical* delusion.

The thirteenth chapter, on the Evolution of Belief, stands in great and remarkable contrast to the remainder of the volume, and the author has felt and excused the inconsistency. It would be impossible to give a more full and frank adhesion to the great doctrine that "all beliefs are the result of the operation of natural causes and of these alone" than the author has here done. "The scientific evidence for this truth (the evolution of belief) is various and overwhelming;" and it is much for the author to admit that this can be said of any scientific evidence. The argument in favour of anti-scientific scepticism drawn from this law of mind is obvious enough. Belief having been evolved from the influences of heredity, environment, and other circumstances, we have reason to expect that it will continue to evolve,—that is to say, to change; and seeing that we have reason to think that the evolution hitherto has been in the direction of truth, we must expect future change to be towards greater truth than we at present possess, which is an admission that what we now believe is true only in part. This argument and its conclusion will, we think, be gladly accepted by those against whom it is aimed, the men of science, for, unlike the philosopher who demands absolute truth in order that he may gain "repose of mind," they covet the changes of extending knowledge, and hoping for nothing more than relative truth, they labour to obtain its continual increase. But it is to be remarked, that although all beliefs result from natural causes, they are not, therefore,

as the author supposes, all within the stream of evolution. There is no reason to expect that any beliefs which can properly be called fundamental, as the belief in one's own existence, will ever change. This, it would seem, must have been the same in kind in the first conscious being as it is now, and, as it would seem, must remain through all the æons of change which may ensue.

We will not follow the author in his latter pages, in which he compares what he calls the two creeds of Science and Religion, and justifies himself for attacking the one because it is, he says, opposed to the other. Should any one wish to avail himself, for a similar purpose, of the weapons he supplies, the best way will be to imitate many novel readers, and commence at the end of the book, which, like the end of a whip, constitutes the lash. Science will perhaps outlive all this destructive criticism, and the humble workers in the pursuit of knowledge who do not uphold any aggressive creed for the purpose of undermining the foundations of faith may take courage from the author's admission made at p. 280: "It is conceded that similar effects always have similar causes, and that a knowledge of particular sequences and co-existences between phenomena can be derived from observation."

The general effect of the book reminds one of the following dialogue in Congreve's 'The Way of the World:'

Petulant. "If he says black's black, if I have a humour to say 'tis blue—let that pass—all's one for that. If I have a humour to prove it, it must be granted."

Whitwoud. "Not positively must—but it may—it may."

Petulant. "Yes, it positively must, upon Proof positive."

Whitwoud. "Ay, upon Proof positive it must; but upon Proof presumptive it only may, that's a logical distinction now—Madam."

Mrs. Marwood. "I perceive your debates are of importance, and very learnedly handled."

Petulant. "Importance is one thing, and learning's another; but a debate's a debate, that I assert."

The degree of proof demanded by our author is Petulant's proof positive, while poor Mr. Mill and others, who diligently hunt the footsteps of Creation, can only with difficulty attain

to proof presumptive. We are glad to find, however, that Mr. Balfour, although he seems quite disposed to expect that others will consider his "piece of criticism so purely destructive in character," more like "a mere dialectical puzzle, a mere exercise in ingenious objections, or even a contribution of a somewhat eccentric kind towards English philosophy," than a serious estimate of the Foundations of Belief, vouches for his own earnestness. The importance and learning of his work may be questioned; but a debate's a debate, and this debate proves with certitude that the author believes in it himself.

JOHN CHARLES BUCKNILL.

The Relations of Mind and Brain. By HENRY CALDERWOOD, LL.D., Professor of Moral Philosophy, University of Edinburgh. London: Macmillan and Co., 1879.

THERE are some who have a vague belief that Physiology is going to supersede Metaphysics, or that all inquiries as to the mode and action of the mind are shrouded in the darkness of the unknowable, and that the utmost we can learn of mental operations consists in ascertaining the swiftness of thought, or the time between a sensation and the answering motion calculated by the help of some delicate chronometrical instrument. But questions connected with the nature and destiny of the human mind are too deeply interwoven with man's dearest hopes and feelings to cease to have an interest for an active and inquiring intellect. Unlike most men who have written on the physiology of the brain, Dr. Calderwood began as a student of Mental Philosophy, and then became a student of Anatomy and Physiology. His book is interesting, not only to the physiologist and neurologist, but also to the philosopher who is anxious to study the relation of feeling and thought to the nervous system.

Dr. Calderwood's reputation as a metaphysician is already established, and in his book he shows that he has mastered what is known of the general anatomy and physiology of the

nervous system down to the very latest researches into the functions of the brain. In his first chapter, "On the Relation of Philosophy and Science," Dr. Calderwood rebuts the accusation made against the Scotch school of philosophy of constructing their theories in disregard of observation. "Those," he writes, "who would fasten upon psychologists of former days such a charge as this must be prepared to maintain that thought and recollection and volition are not facts, or that these are facts which do not occur according to law, or that both facts and laws are beyond the reach of scientific inquiry." Dr. Calderwood has little difficulty in showing the fallacy of Dr. Maudsley's attack on the trustworthiness of consciousness. "It has no doubt been said that there 'is no agreement' among those who resort to internal observation, but every one who is familiar with the works of psychologists knows that the statement is inaccurate. As to the laws of observation, of association, of reasoning, of pleasurable feeling, there is all but perfect agreement among them." And again: "It were indeed strange were any science to offer itself as defender of the dogma—that man is not to trust his own experience. What may he trust if not this? Take the most ordinary and commonplace tests. How but by consciousness does any man know that the blue appearance of his hands on a wintry morning is the accompaniment of coldness, or that perspiration is attendant on a high degree of heat? When a man says that he feels warm, is it a sad misfortune that 'he appeals to a witness whose evidence can be taken by no one but himself?' Is it true that this is a witness 'whose veracity cannot be tested?' To throw doubt upon experience is to deny the possibility of knowledge, and so of all science. When the psychologist trusts his consciousness he simply does what every man does—what every man must do, who is not insane. Trust in consciousness is no speciality of psychologists, but the common prerogative of humanity."

Dr. Calderwood then proceeds, in four chapters, comprising about 150 pages, to give a sketch of the comparative anatomy of the brain and nervous system, and to discuss what is known of their functions. These chapters, though of course compilations, are written in a shrewd and critical vein, which displays

the author's vigorous train of thought. Working with the assistance of such distinguished scientific men as Professor Turner, Dr. Rutherford, Dr. Grainger Stewart, and Dr. Clouston, this part of the work may be regarded as a trustworthy *résumé* of the newest results of modern investigation into the functions of the nervous centres, written in a style which renders it of easy understanding to any cultivated reader. The descriptive portion of the book is illustrated by twenty-eight engravings on wood, taken principally from published works.

In Chapter V. he compares the brain and intelligence of man with those of some of the higher animals, especially the dog, horse, ape, and elephant. Dr. Calderwood places the intelligence of the dog much higher than any of the others, and quotes authorities to show that the sagacity of the elephant has been much overrated. He shows that while in the anthropoid ape there is a near approximation to the human brain, the creature does not show intelligence equal to that observed in the ordinary life of animals, much less similar in the structure of the encephalon.

"The ape is far less like the man in intelligence than its brain is like to the human brain." He reaches the conclusion that the brains most elaborate in convolution are associated with the most highly developed muscular system, and finds in the similarity of the body in the ape the explanation of the similarity of its brain.

Dr. Calderwood does not seem, at p. 206, to distinguish nicely between complexity of the muscular system admitting of diversity of execution, and mere mass of muscles allowing for great exertion of force. The brain of the horse, the elephant, or the whale, is much more finely convoluted than that of the cat; but yet the cat is assuredly much more capable of fine and difficult motions than the horse or whale; and although the elephant is an animal of colossal stature, its strength seems to me to be feeble in comparison with its bulk. Dr. Calderwood might advantageously have considered the case of the seal, an animal with a well-developed brain and possessing a good amount of intelligence, but very limited power of muscular exertion.

In general, Dr. Calderwood takes a much lower view of the

intelligence of animals than naturalists who think that the human intellect has been evolved by a series of improvements from that of the brute.

He doubts whether animals can exercise any voluntary determination over their actions, and thinks that most things that they do can be accounted for by sensori-motor activity. Dr. Calderwood keeps clear of the question whether the mental manifestations of the higher vertebrate animals are to be accounted for by the hypothesis of a supersensuous principle, or are simply a form of nervous activity. However low we may rate the amount of reason in the lower animals, most people will be disposed to admit that if what thought exists in the dog is but a function of the brain, even the highest abstract thought might be a function of the brain in man. Hence naturalists like Agassiz, who believe that there is a soul in man, admit the existence of a similar immaterial principle in animals. We should like to have Dr. Calderwood's opinion on this point, but the learned professor is too skilful a dialectician to give battle save on ground selected by himself.

When we come to Chapter VII. "On Personal Experience as connected with Sensation," we feel the advantage of Dr. Calderwood's skill as a metaphysician. He does not contest that molecular changes accompany sensation, motor impulses and thought, but he shows that there is no logical connection recognised by the mind between motions in cells and thought and sensation. "All that we know of nerve-substance," he writes, "whether fibre or cell, fails to explain the simplest and most familiar fact in our experience." Not only have we a series of sensations, but we recognise this series, and assume a personality which has experienced those that are past.

Dr. Calderwood makes use of the argument so well handled by Huber, in his tract, 'Ueber das Gedächtniss:' "Even if," he observes, "physiological hypothesis were ventured in the form of a suggestion that there may be in the sensory cell a *register* of the shock delivered there, this would not help us towards an explanation of the facts of consciousness. Even if there were such a register, and the registration were made on a sensitive surface, and were permanent, this would not meet the requirements of the case. A register contains the mate-

rials for comparison, but does not institute comparisons. The facts carry us quite beyond mechanical contrivance, inasmuch as one thing not only follows another, but one thing is compared with another; that is, there is not only one thing distinct from another, but one thing is distinguished from another."

Dr. Calderwood takes up a familiar calculation of so many cells to the square inch, and gravely argues that there is not a sufficient number of cells for the vast number of ideas which we acquire. We have always considered this sort of thing so much solemn trifling; as Spinoza observes we cannot conceive of a space bounding an idea, or an idea bounding a space. Could a big nerve-cell hold more ideas than a small one? Or does a great idea ever smash a frail one, if it comes to a collision in a nerve-fibre?

Dr. Calderwood goes on mingling metaphysics with physiology and cerebral pathology, as far as they may be mingled. He treats of the use of speech, the action and reaction of mind and body, sleep and unconsciousness. He tries to show how the observations of physicians in insanity do not invalidate the belief in the independent existence of the human mind. This part of his argument is treated with great care, and shows considerable reading and fulness of information.

It is no doubt the duty of a critic to pick out a number of faults and oversights in the book, and I am concerned to think how carelessly this office must have been performed, for I cannot recall any salient blunders, and am not willing to read the book over again. Now and then there is a certain simplicity noticed which bespeaks the amateur, as when Dr. Calderwood quotes Carpenter's 'Physiology,' to prove the fact familiar to every surgeon, that a severed nerve may be reunited, and the power of communication restored; or when he speaks of checking brain-wasting, and reinvigorating the organ with "such things" as tincture of opium and sulphuric ether.

Dr. Calderwood's idea that we may have loss of a portion of brain-substance with advantage to the mental powers is perhaps original; not so his suggestion that the brain development may so far naturally exceed the capacity of the cranium as to cause compression of the whole brain. His medical

friends will be able to put in his hand some account of the disease called hypertrophy of the brain.

In his concluding Chapter, "On the Higher Forms of Mental Activity," Dr. Calderwood lays great stress upon the existence of a sense of duty in man, and a sense of dependence upon a higher power which he thinks could not have originated from any organisation of matter; but for this and other arguments the reader must turn to the book itself.

Enough has been said to show the character of the work. The subject must be an interesting one to all who occupy themselves with mental philosophy, and it is treated in a manner worthy of its importance.

WILLIAM W. IRELAND.

Müller on the Acute Anterior Poliomyelitis of the Adult. (*Die Acute Atrophische Spinallähmung der Erwachsenen; eine klinische Studie.*) By DR. F. MÜLLER. (Pages 105.) 8vo. Stuttgart: Enke, 1880.

FEW clinical types have been more diligently and successfully studied during the last decade than that originating from an inflammation, acute or chronic, of the anterior horn of spinal grey matter. "Poliomyelitic" diseases comprise the "essential" or infantile paralysis of the older authors, the (acute or chronic) atrophic spinal paralysis of the adult, bulbar paralysis, and some forms at least of progressive muscular atrophy. The name "regressive paralysis," which has been proposed, seems unfortunate, for though expressive enough of some characteristic features of the acute forms, it certainly fails to apply to the subacute and chronic forms.

The first author who seems to have had a clear idea of the "infantile" type is Underwood (1784), who in his work on diseases of children describes it as paralysis "of dentition." Kennedy (Dub. Med. Jour. 1841) speaks of a "temporary" paralysis of children, and the name "essential" was given to the disease by Barthez and Rilliet. It was with Heine and

Duchenne (1850–1860) that the idea of a spinal localisation arose, and their theoretical assumption has since been verified by numerous autopsies.

The clinical characteristics of the infantile disease, as delineated by Duchenne, were so striking as not to fail being recognised in a number of adult cases; and it was about ten years ago that the indefatigable author of the 'Electrisation Localisée,' described a "Paralysie spinale antérieure aiguë de l'adulte, ou par atrophie des cellules antérieures." Dr. Müller, in the able and exhaustive monograph, gives us a careful *résumé* of all the cases published from Duchenne's to the present day, and adds four observations of his own; one of them quite unique, owing to the fact that the patient was under observation from the very beginning of the disease; and that a most thorough electrical exploration of nerves and muscles was carried out from the very onslaught of the paralytical symptoms daily during the first week, and at appropriate intervals during eighteen months subsequently.

The author holds that *fever* is present in all cases as an initial symptom. It can be only a "lapsus calami" of Eulenburg's, when he says that "fever has hitherto not been observed" in the disease (Lehrbuch, vol. ii. p. 378). From the cases hitherto published it would appear that the duration of febrile disturbance is usually from two to seven days. The intensity of the fever varies much, but bears no proportion to the extent of the subsequent paralysis.

Pain is a very common symptom during the first stage of the disease, and is usually of a tearing character; but, like fever, varies much as to duration and intensity. It extends over the limbs, the sacrum, or along the vertebræ. It disappears with the invasion of the paralysis. It must be noted, however, that later on other algesic symptoms may supervene, which, however, are duller in character and more localised in the muscles undergoing atrophy.

Various *paræsthesiæ* are apt to make their appearance; but the presence of any anæsthesiæ at once relegates the case from the poliomyelitic group to that of the more diffuse myelitis, where the posterior tracts of the case are involved; as, for instance, in the case described by Glynn ('Lancet,' 1878,

vol. ii. p. 395), where there occurred a complete anæsthesia of the four extremities, lasting two months.

The leading symptom, *loss of motor power*, occurs as a rule abruptly, not gradually. Whereas in the infantile form monoplegic lesions are the most common, in the adult the four extremities are most usually affected; each occurring in about 50 per cent. of the respective cases. An interesting point, to which I shall have the opportunity of referring more fully elsewhere, is that the paralysis does not distribute itself among muscles according to their peripheral nervous supply, but rather according to their central nuclear connections. No muscle can claim immunity from poliomyelitic processes, though some writers have expressed themselves in this sense. Both tendon and skin *reflexes* are, as a rule, abolished or impaired, but reappear in the regressive stage, though apparently without keeping pace with the rate of recovery of the muscular power.

In typical cases of poliomyelitis, no vesical, nor rectal, nor genital disturbances occur. On the other hand, *vaso-motor* changes are always present. Dr. Müller found in his case that the lividity and coldness of the affected extremities which characterise the later stages, are preceded during the first few days by a hyperæmic and hyperthermic condition.

From the fact that disturbances in the functions of the sweat glands are but rarely observed in the paralysed limbs, Dr. Müller concludes that their functions are independent of the vaso-motor apparatus, which is always impaired; and that the nervous centres which govern them are not situated in the motor tract of the anterior horns.

The only *trophic* troubles present in poliomyelitis consist in degenerative changes of nerve and muscle, as evidenced by the frequent wasting of the limb and by the electrical reactions obtained. The latter have been, as before mentioned, most fully investigated in one of Dr. Müller's cases; and his results concord most satisfactorily with the phenomena of the "reaction of degeneration," so ably described by Professor Erb in vols. xi. and xii. of Ziemssen's 'Cyclopædia.' The facts may be summarised as follows: The farado-nervous excitability disappears rapidly; in the most severely affected nerves

within five or six days of the paralysis. The time of its reappearance varies much, and is always preceded by that of voluntary stimulation. In permanently disabled nerves it, of course, vanishes for ever.

The galvano-nervous excitability follows closely the behaviour of the latter, but takes about two days longer to disappear. Its restoration also follows that of voluntary power.

There are frequently a number of nerves which, though the muscles supplied by them may undergo atrophy, and all the changes of reaction dependent upon it, never show any, or but a slight, diminution in farado-galvanic excitability. Cases where this occurs form an interesting transitional group, characterised by a "partial" (or "middle") form of reaction of degeneration, which shows a disturbance in the spinal influence on the nutrition of the muscle only, and not of its supplying nerve.

With regard to muscles, their farado-excitability diminishes more slowly than that of the nerves, but eventually disappears completely. But the most interesting changes are those offered by the galvano-muscular reactions. After undergoing a slight diminution (which in Dr. Müller's case lasted about three weeks), the galvano-muscular excitability rises, and remains above the normal for a few days; then it gradually diminishes again, whilst the pathognomonic alterations in polar effects manifest themselves. These, in severely affected muscles, amount to a complete inversion of the normal polar formula: A.C.C. > C.C.C., and C.O.C. > A.O.C. In muscles whose degeneration is less marked, we get A.C.C. = C.C.C., or simply a slight rise of the anodal effect, without any corresponding cathodal increase. At the same time the mechanical irritability of muscles increases, and the contractions are altered in character, becoming protracted, sluggish, vermiform. These alterations are of great prognostic importance. Muscles in which recovery is never to take place, have a uniform downward progress; stronger and stronger currents are necessary to obtain the trace of a contraction, the "ultimum moriens" being the A.C.C. In less severely affected muscles the qualitative alterations of polar reactions are not so marked at first,

and gradually return to the normal formula as the degenerative process enters into a regressive stage. Of course the partial form of degenerative reaction, viz. where the nerve does not share in the morbid process, is of far better augury as to the eventual recovery.

An interesting fact is mentioned by Dr. Müller. The paralysed right rectus abdominis showed no quantitative nor qualitative departure from the normal reactions; apparently a contradictory result. I might suggest as an explanation that, as in some forms of peripheral paralysis (such as mild cases of facial or musculo-spiral paralyses), where the electrical reactions remain normal throughout, the transmission of voluntary impulses alone is interfered with by the lesion, whilst that of trophic influence is left intact. This is made all the more admissible by the well-known facts that in mixed nerves sensibility resists influences destructive of motility; and that, as we have just seen, muscle-nutrition may be affected without any participation of the nerve in the morbid process.

The *regressive stage* of the disease is inaugurated by the restoration of those muscles in which mere quantitative electrical changes have been observed. This may begin as early as within a week of the paralysis; or may be delayed for two or more months when the lesion is more severe. The atrophy of the muscles may be masked by deposition of adipose tissue. As a rule, residual paralyses persist here and there, and secondary contractures may supervene. With regard to the *morbid anatomy* of the acute poliomyelitis of the adult, it is only since Schultze published his case (Virchow's 'Archiv,' 1878), that a complete answer could be given to those who objected to the localisation of the disease in the anterior horns. The patient, aged 40, died twenty months after the attack.

Macroscopically, two spots of the anterior horns in the left cervical and right lumbar enlargement were found discoloured; and the horn there diminished in diameter. These spots, microscopically examined, showed a nearly complete disappearance of the ganglionic cells and nervous elements between them; a thickening, with nuclear proliferation, of the neighbouring blood-vessels; the intra-medullary portion of anterior roots, with but few, thin, axis-cylinders. Similar,

though much less marked changes existed throughout both anterior tracts of grey matter. The posterior portions of the cord and antero-lateral columns presented nothing abnormal, but an increased size of some axis-cylinders at one or two points. In the sciatic nerves some bundles of fibres were found converted into tracts of connective tissue.

The usual degenerative changes were observed in the paralysed muscles. Respecting the *pathogeny* of the disease, Dr. Müller points out that, owing to the physiological and histological peculiarities of the anterior horns, as well as to the marked independence of their capillary plexus, the possibility of their being seized upon by a pathological process, with but little implication of the antero-lateral and posterior tracts, is at once made evident. Then, again, the symptoms, both negative and positive, agree so well with what is known of the localisation of functions in the cord, that we feel justified in calling the disease a poliomyelitis. Lastly, such a case as Schultze's, where the whole length of both anterior horns was shown to have been the seat of inflammatory phenomena, with next to no lesion of the rest of the cord, confirms our present theory of the disease.

The *etiology* is yet obscure. With regard to age, the third septennium seems to be the most favourable for the development of the disease; the age of 16 out of 47 patients ranges between 14 and 22. Out of 40 cases, 19 occurred in August–September; January shows 7 cases, the remainder being distributed over the other months. As to sex, males form nearly two-thirds of the patients. As in many other diseases, exposure to cold seems to act as exciting cause in adult poliomyelitis. In 25 out of 47 cases this was emphatically the case.

The *diagnosis* of the typical disease is easy. Dr. Müller, however, fully discusses the differentiation of those cases where mistakes might be possible; but our space prevents our following him in the interesting details he gives. The *prognosis*, "quoad vitam," is favourable, and the course has been sufficiently made clear in discussing the electrical condition of the muscles. With regard to the *treatment*, the author is very partial to hypodermic injections of ergotin, with atropia during the hyperæmic stage. Afterwards a tonic regimen, with iodide

of potassium and wet-sheet packings, to stimulate the resorption. The paralytical symptoms have often been combated with strychnia. But we agree with the author in rejecting this drug as useless in poliomyelitis. The only chance we possess to hasten and extend the natural process of restoration is the rational and persistent use of electricity. The treatment must be begun very early, and carried on patiently for months, nay years, if necessary. The best plan is to apply the positive pole (using a *large* plate; the usual sponge-holders are worse than useless) to the cervical or lumbar enlargements, for about five minutes, whilst the negative is placed on the affected muscles. A few interruptions, or voltaic alternatives, are then to be made in order to bring about contractions. Voluntary exercise, and promoting the warmth of the limbs, especially by massage, are valuable auxiliaries.

We recommend Dr. Müller's monograph to the attention of our readers, and thank him for his solid contribution to the pathology of the spinal cord.

A. DE WATTEVILLE.

Clinical Cases.

FIVE CASES OF DISEASE OF THE BRAIN, STUDIED CHIEFLY WITH REFERENCE TO LOCALISATION.

BY CHARLES K. MILLS, M.D.

Neurologist to the Philadelphia Hospital.

I. Softening of the Pons Varolii.
II. Tumour of the Pons Varolii.
III. Localised Tubercular Meningitis.
IV. Localised Tubercular Meningitis; Internal Pachymeningitis.
V. Multiple Cerebral Embolism.

THE cases contained in this paper are, I think, of value from a number of stand-points, but my remarks upon them will be brief, and will chiefly refer to their bearing upon the question of cerebral localisation. Much of pathological interest is taught by them in regard to such subjects as thrombosis and embolism, necrotic and inflammatory softening, meningeal tuberculosis and pachymeningitis, and primary and secondary disease of the cerebral vessels; but too elaborate a discussion of separate features would have extended the article to unwieldy limits. Even with reference to localisation, the cases are, as far as possible, allowed to speak for themselves. Cases of multiple or diffused lesions, such as three of the five here presented, are not, as some suppose, without value in the study of the problem of the cerebral functions. If, for instance, we have a symptom-picture, which points with great positiveness to lesions of the cortical motor zone, and if, after death, such lesions are found, we should not disregard them altogether because of complicating conditions. In every field of medicine we are compelled to resort to processes of

comparison and exclusion, in order to arrive at a satisfactory explanation of special manifestations. The all-important point is that we shall be honest and thorough in our investigations.

CASE I.—*Softening of the Pons Varolii—Right Hemiplegia followed by General Paralysis—Rapid Elevation of Temperature before Death.*

B——, aged 40, was admitted to the Philadelphia Hospital on May 20, 1879. He stated that three weeks before, while lying down, he suddenly felt as if his head was spinning round. He tried to get on his feet, but could not do so on account of the vertigo and a general loss of strength. He did not lose consciousness during the seizure. His speech became so "thick" that he could not be understood, and he found, on rallying from the first effects of the attack, that he was partially paralysed in the right arm and leg, and that his face was a little drawn to the left.

For several weeks previous to this seizure he had suffered with headache. He had abused alcohol, and also had a history of syphilis.

During the three weeks previous to his admission he had been slowly improving. He was hemiplegic on the right side, and was weak in all his limbs, but he could sit up and could walk around, holding on to the beds and chairs with his left hand. The paralysis was more decided in the right upper extremity than in the lower, but it was well marked in both. He had a slight left convergent strabismus, and his mouth was drawn a little to the left, but his face was not otherwise affected; no ptosis, external strabismus, lagophthalmus, or other symptoms, except those noted, being present. He could converse without difficulty, although his speech was not perfectly distinct, owing apparently to slight lingual and oral weakness. He had full control of rectum and bladder. Sensation was well preserved. He was pale and haggard.

The above was his condition during the first twenty-four hours after coming under observation on the 20th of May. On the 21st, he called the attention of the nurse to the fact that he had suddenly become paralysed in the left arm; during the same day his left leg was attacked; he became much weaker in every respect, and had to take to his bed. He did not change notably in his symptoms until the 23rd, when he began to complain of severe frontal headache. On this day his urine was examined, and was found to contain neither albumen, tube-casts, nor sugar.

On the 24th he had a peculiar seizure, which, as I chanced

to be present in the ward at the time, I was fortunate enough to witness. His face was pale and anxious, and he broke out into a profuse general perspiration. The left internal strabismus was observed to be much more marked than it was on admission. Respiration became difficult, but it was not stertorous; he breathed in a jerky manner, and was almost constantly making a puffing sound with his mouth, at which also he frothed a little. He could not speak, and could not thrust his tongue beyond his lips. On talking to him, he seemed to understand all that was said to him, and would make futile efforts to reply; he would become highly excited, looking at those around him with a most beseeching expression, while the tears suffused his cheeks, as if appreciating the helplessness and hopelessness of his situation. He could only open his mouth enough to separate his teeth from a quarter to half an inch. Liquid food and medicine could be taken, but he had some difficulty in swallowing, owing to a tendency to regurgitation. He was helpless in all his limbs, but the loss of power seemed still more prominent on the right. Slight flexions were present at the wrist, in the fingers of the right side, and at both knees. At intervals he had considerable convulsive tremor, affecting especially the right side, and the leg more than the arm. His pulse was 88 and weak; his temperature, taken in the rectum, was 100·4° F. Involuntary passages did not occur; nausea and vomiting were not present. His eyes and neck, at this time, showed no tendency to deviate either to the right or left.

On the 25th, 26th, and 27th, he continued in nearly the same condition, having improved slightly, under counter-irritation to the back of the neck and extremities, and the use of stimulants. He still perspired freely nearly all the time; he remained anxious-looking and emotional, his breathing became a little better, but continued to be of the same puffing, jerky character; he did not regain the power of speaking, thrusting out his tongue, or swallowing without difficulty; the strabismus and helplessness of his limbs did not change. His pulse was weak, and ranged between 100 and 112. His temperature, taken in the rectum, varied but little during these three days. The record was as follows:—

	Morning.	Evening.
May 25	100° F.	Not taken.
„ 26	100° F.	100·4° F.
„ 27	99·8° F.	100·3° F.

On the morning of the 28th he became much worse. Sweating became more profuse; his paralytic symptoms deepened;

about midday involuntary discharges took place; his head and eyes, for the first time, showed a constant tendency to turn to the *right;* his breathing became more and more irregular and difficult. His temperature shot up rapidly; at 7 A.M. it was 101·3° F.; at 3 P.M., 105·5°; at 6½ P.M., 108°; after this it was not taken. He died at 11½ P.M.

An autopsy was held fourteen hours after death. The pia mater exhibited slight general cloudiness, with scattered patches of extreme redness. On examining the base the basilar artery was found to be enlarged to nearly twice its usual diameter, its walls being thickened and rigid, and rough on the outside. Some of the small vessels, which go down from it into the pons, could be seen to be obliterated; the basilar trunk itself, however, was quite free and of good calibre, and was readily opened with the scissors. The other main arteries, and their primary and secondary branches, showed wide-spread evidences of atheroma.

The pons varolii was the seat of an extensive and interesting lesion, the following description of which is partly from notes made at the time of the post-mortem examination, and partly from careful subsequent investigation of the specimen. This lesion was an irregular area of softening, the centre of which was just below the centre of the pons. Superficially, the softening was an inch in greatest length, which was from above downwards and to the left of the median line, and three-fourths of an inch in width at its widest part, which was near its lower boundary. Vertically, the softening extended about one-fourth of an inch higher to the left of the median line; while horizontally, and below the centre, it reached about one-eighth of an inch farther to the right than to the left. It presented four spots of excavation, the intervening spaces being filled up with softened and broken-down tissue; one of these was a comparatively large central space, the second was to the left and above, the third to the left and below, and the fourth to the right and below the centre. The entire surface-softening was included in an irregular triangle, formed by joining these outer points of depression or excavation. On subsequent close examination, the central region of the pons was found to be scooped out to the depth of half an inch, and it was also irregularly invaded in all directions by softened tissue; but its upper and lower fifths, and lateral bands of from one-fifth to one-sixth of its width, were unaffected. The cranial nerves, superficially, were not involved. The floor of the fourth ventricle presented a healthy appearance.

An extravasation as large as the palm of the hand was present in the cardiac end of the stomach. The aorta was

atheromatous. Both kidneys were fatty, the degeneration being more advanced in the right than in the left.

Remarks.—Summing up this case, the chief points are found to be as follows: headache; a vertiginous seizure, which left partial right hemiplegia; left convergent strabismus; defective articulation; paralysis of the left arm coming on suddenly three weeks after the first attack, but without any special head or general symptoms; a third seizure, accompanied by pallor, anxiety of expression, great emotionality, profuse perspiration, difficulty in breathing and swallowing; inability to speak, to thrust out the tongue, or open the mouth widely; paralysis of both arms and both legs, but still more pronounced on the right; convulsive tremor, most marked in right leg; weak and frequent pulse; slight elevation of rectal temperature for four days. On the day of death, deepening of all the symptoms just noted, and, in addition, paralysis of bladder and bowels, conjugate deviation to the right, and rapid elevation of temperature to 108° F. Some interesting negative facts are, absence of anæsthesia, of nausea and vomiting, of true convulsions, and of changes in the urine.

At first sight this case might be looked upon as contradictory of the views of those who hold to the decussation at the anterior pyramids of voluntary motor fibres, but I doubt whether it should be so regarded. Leaving out the question of some possible peculiarity in the decussation, in regard to which I have not yet had the opportunity to determine positively, we have in the history of the case, conjoined with the results of the autopsy, a satisfactory explanation of all the manifestations. The paralysis following the first seizure was of the face and limbs of the right side, and it continued until the day of death to be most decided on the right. The autopsy revealed an area of softening in the pons, occupying the centre and both sides of the median line, and this was evidently due to closure by a thrombotic process of some of the little nutrient arteries given off from the basilar. The different vessels probably became obstructed at intervals corresponding to the several successive seizures which the patient experienced. The softening in the centre and to the left may, in this way, have taken place earliest, an order of events which would best account for the right hemiplegia. The spots of excavation, spoken of in the notes of the autopsy, possibly represented the point at which the successive attacks of softening occurred. Paralysis of both sides of the body did occur before the death of the patient, as might be expected from a lesion of the kind and in the situation described.

Convergent strabismus, inability to articulate, to thrust out

the tongue or to open the mouth widely, and difficulty in swallowing and breathing, as the cranial nerves were not affected superficially, point to the probable involvement of the nuclei or deep tracks of the abducens, facial, hypoglossal, motor-trigeminal, glossopharyngeal, pneumogastric and spinal accessory. The escape of the posterior columns and the floor of the fourth ventricle are of interest in connection with the absence of anæsthesia, of nausea and vomiting, and of true convulsions.

The temperature observations made upon this case, although limited, are sufficient to be of some value in reference to the question of a heat-centre in the pons. Supposing the existence of such a centre, it is probable that the first foci of softening did not implicate it directly, but as the necrotic area enlarged, this centre became eventually involved.

CASE II.—*Tumour of the Pons Varolii—Convulsions, Vomiting, Rotatory Movements towards the Left—Final Hæmorrhage into the Medulla Oblongata.*

J——, 35 years of age, seven years before coming under observation, began occasionally to have headaches, and a year later became subject also to temporal and orbital neuralgias. The headaches and neuralgic attacks gradually grew worse and worse, both as regards frequency and severity. Five years after his first headaches he began to have "spells," in which he would be unconscious for a moment or two, and would have twitchings about the mouth and in the hands. These seizures returned at intervals of from one to two months for about a year, when they were supplanted by general convulsions. Sometimes he would have several convulsive paroxysms in the same day. Before the attacks and during these days of spasmodic storm he suffered with terrible headache and uncontrollable vomiting. Often at other times, without convulsions, he had spells of nausea and headache. Ophthalmoscopic examination showed atrophy of both optic nerves. His memory had failed, and he was usually in poor spirits. No positive paralysis was ever observed, although he was never closely examined for slight deficiencies in strength. His bowels were obstinately constipated, and he suffered nearly all the time from fæcal accumulations. He occasionally had "cramps" in the stomach and legs. He denied venereal disease of any kind, but his wife had had several stillborn children. The bromides, potassium-iodide, mercurials, and measures to relieve special symptoms, such as constipation, were employed, but without doing more than slight temporary good.

Six weeks before his death I saw him in one of a series of terrible convulsions. Although the convulsion was general, his right side seemed to be more affected than the left. Lying on his back, the spasms had the effect of lifting up the right side of his body and causing him to work over towards the left, as if trying to get on his face in this direction; but before getting altogether on his left side, he would fall backwards again, when the same curious lifting and rotary movement would be repeated. Frothy saliva escaped from his mouth; his face was much contorted, and was pale and livid by turns. The seizure lasted in all three minutes; when he relaxed he was bathed in perspiration and went off into a stertorous sleep. He died at the close of a series of similar convulsions, his death being preceded by general paralysis and involuntary evacuations.

The post-mortem examination, made twenty-three hours after death, revealed a round white tumour, a quarter of an inch in diameter, in the pia mater just to the left of the centre of the anterior surface of the pons varolii. The entire anterior central portion of the pons was softer and more doughy than usual. Just at the junction of the pons with the medulla oblongata was a recent hæmorrhage, which had spread downwards to about the middle of the latter. No other lesions of the brain were discovered, and this was the only organ examined.

Microscopic examination showed the tumour found in the pia mater to be a granulation tissue, probably of syphilitic origin. Nothing but mere detritus masses could be detected in the softened portion of the pons, and it could not therefore be determined positively whether this was a broken-down gumma.

Remarks.—The important symptoms in this case were headache, neuralgia in a portion of the trigeminal distribution; *petit mal*, supplanted after a time by fully formed convulsions; convulsions general, but the movements more marked on the right half of the body; during the convulsions, execution of a rotatory movement towards the left; failure of sight and optic atrophy; loss of memory and depression of spirits; frequent spells of nausea and uncontrollable vomiting at the time of the seizures; obstinate constipation, and occasional cramps or spasms in the stomach and legs. The above are points carefully studied and noted when the observations were made. The case, however, was under my care several years since, when I was not so fully impressed as I am now with the importance of investigating minutely. Sensation was not

closely studied, although I remember distinctly that on several occasions the patient complained of numbness of one side of the face. No definite paralysis of face, arms, or legs, of muscles or muscular groups, was present, although the man showed general and gradually progressing weakness of all the limbs. The trigeminal neuralgia, with probable anæsthesia, the peculiar epileptiform seizures, the local cramps or spasms, and the excessive vomiting, were symptoms which pointed to tumour of the pons—to a strongly irritative lesion in this region. The headache, optic atrophy and mental failure are the accompaniments of cerebral tumours in various locations. General weakness of all the limbs is the condition found in a number of cases of central lesion of the pons. Even supposing the central softened mass on the anterior surface of the pons to be a gummatous tumour, some of the fibres constituting the motor tracks on both sides were probably uninjured. The peculiar rotatory movements towards the left, recall the experiments in which Schiff divided the middle cerebellar peduncles at the side of the pons, the result being the performance by the animal of a rotatory movement towards the side of the lesion. The pial tumour was a little to the left of the median line, although the actual destruction of the pons was central. The fibres which go to form the middle cerebellar peduncle were, no doubt, affected by pressure and directly, the irritation being greatest to the left. The lesions were so situated as not to have involved directly the great sensory tracts. The hæmorrhage which spread over the medulla oblongata was doubtless the immediate cause of death; and this was probably brought about by the impairment of the vessels by disease of their walls and by proximity to the growth, and by the rise in blood-pressure during the terrible epileptiform paroxysms.

CASE III.—*Localised Tubercular Meningitis, with Cortical Softening—Involvement of the Centres for the Limbs and Face.*

M. H——, 30 years of age, could not give a very clear and full previous history; but stated that one year before coming under observation she had been ill for several weeks; that she had had high fever, with great pain in her limbs, particularly in the right arm and leg. During and after this attack she suffered almost constantly from headache, and occasionally from dizziness. She had also been troubled with cough. Four months before her admission to the hospital, while standing over a fire cooking, her right arm suddenly fell helpless

to her side. About ten days later, the right side of her face became partially paralysed; and still later, but exactly when she did not remember, her right leg became weak. Two months before admission she had an inflammatory affection of the right eye, from which she had just recovered. She had kept on her feet most of the time, but says that she was feverish and weak, and was much worried with pain in her head.

On examination I found that the right arm and right leg, which were about equally affected, were markedly weaker than the limbs of the left side; they were decidedly paretic, but presented no contractures, and only slight wasting. The lower part of the right side of the face was also paretic, as was shown by slight dropping of the nostril and corner of the mouth, and twisting to the left of the mouth. She could not draw up the right angle of the mouth when directed to do so, as she could the left. The tongue was not deflected, but its movements were performed in a weakly, tremulous manner. The right eyelid drooped slightly. No strabismus was present. The condition of the pupil could not be determined, on account of an opacity which covered the right cornea almost entirely, and was the result of the recent keratitis from which the patient had been suffering. Both eyelids could be opened and closed, and frontal furrows were present on both sides. Her manner of speaking was hesitating and unsteady, as if from weakness of the oral and lingual muscles. She was not aphasic, in the sense of having any partial or complete loss of speech. She did not drop letters, syllables, or words; but talked along, without stopping, in a drawling, uncertain, slipshod sort of way. Sensation was unimpaired. Hearing was good. Smell was defective, but not lost on either side. She could not, of course, see with the right eye, with its opaque cornea; the sight of the left was poor. Ophthalmoscopic examination, by Dr. E. O. Shakespeare, revealed a beginning neuroretinitis. Her mind acted torpidly, and, judging from the results of questioning her in regard to her past history, her memory was of the poorest kind. She had full control of her evacuations. She had no history of vomiting or convulsions. Her appetite was poor, and she was constipated. No examination of pulse or temperature was at this time made. She had probably had venereal disease, but I could not determine this certainly. Percussion and auscultation showed partial consolidation of the apices of both lungs.

She was ordered potassium iodide and cod-liver oil. Sinapisms were also applied to the neck, and a calomel purge was given occasionally. She improved a little in every respect;

but particularly as regards her headache, which almost entirely left her. After remaining under treatment for five weeks, during which time she was never confined to her bed, she asked for her discharge, and left to return to her family; but twelve days later she was again brought to the hospital, but now in a helpless and semi-conscious condition, from which she never rallied, dying one week after her re-admission. A few points were again noted. The loss of power in the right arm and leg had become a marked paralysis. The leg could be moved up and down over a space of a few inches, as she lay in bed. The paresis of the lower facial fibres was more positive; and both ptosis and rotation outwards of the eye were present and well marked. At intervals the right leg, and the arm and leg of the *left* side were the seat of twitching or convulsive tremor. She had fever, with paroxysms of sweating; she was delirious at times; her pulse was frequent and irregular.

The autopsy was held sixteen hours after death. The dura mater of the left hemisphere was slightly adherent in its inner surface at two points along the parietal margin of the fissure of Rolando, one at the median end of the fissure and the other just above its centre. On pulling the membrane away, which could be easily done, a mass of soft yellow exudation was found at each place of adhesion. The pia mater, both anterior and posterior to the fissure of Rolando, presented evidences of inflammatory action, being more or less opaque, streaked, and hyperæmic. On attempting to remove it, at several points its under-surface was found to be adherent to the convolutions, and to be the seat of what appeared to be tubercular masses, with purulent exudation, the cortex being superficially softened at some of the points. The peculiar character and localisation of the pathological process in the pia mater and cortex may be understood from the following description: (1) The pia mater was opaque, thickened slightly, and adherent at points over the paracentral lobule and the upper extremities of both ascending convolutions, the cortex being softened over a spot one-fourth of an inch in diameter in the ascending parietal convolution, at its upper limit. (2) Deposits, yellowish exudation, and a space of superficial softening, half an inch in diameter, were present about the centre of the ascending parietal convolution. (3) Two foci of cortical softening were found in the ascending convolutions, on each side of the lower fourth of Rolando's fissure, with pial changes at corresponding points.

The sides of the fissure of Rolando, at its middle third, were bound together by inflammatory products.

The pia mater of the interpeduncular space was very opaque,

much thickened, and of a gelatinous consistence; and the changed condition of the membrane, but with less opacity and thickening, continued into the left fissure of Sylvius, and upwards from the horizontal branch of this fissure along the convexity of the hemisphere in the general region of the distribution of the Sylvian artery. Greyish nodules were found here and there along the vessels, and a few purulent streaks and hyperæmic spots, besides the special areas along the fissure of Rolando already described. The pia around the optic chiasm, and particularly on its left side, was markedly thickened and changed in appearance. All the vessels, nerves, and bodies, at the base, from the pons to the chiasm, were more or less involved in an inflammatory process of the membrane. The microscopic appearances pointed to a tubercular inflammation of the pia mater, confined chiefly to the central poration of the base of the brain, and to the territory of the left middle cerebral artery. The tubercular process had been most extensive, and its effects most severe, at the points along the fissure of Rolando, and over the paracentral lobule, which I have specifically described. Some development of miliary tubercles had probably occurred at points in the cortex itself. Small vessels had certainly been obliterated. The pia mater over the pons and medulla, over both occipital lobes, and along the edges of the longitudinal fissure, was much more injected than it is usually found, but no tubercular appearances were discovered outside of the territories detailed. The ganglia, and all portions of the brain, were examined, but no other abnormal conditions were found. I was not able to secure an examination of any other organs.

Several specimens of the diseased pia mater, with blood-vessels, and the softened cortex, and the eyes with parts of the optic nerves attached, were submitted to Dr. E. O. Shakespeare for microscopical examination, and to him I owe the following valuable report, and also the opportunity of examining the mounted specimens:—

"The specimens of the membranes and cortex of the brain, and of some of the larger cerebral vessels, were found in good condition for microscopical examination. Thin sections were made from the softened cerebral convolutions, with its attached pial covering, were stained with carmine, and were temporarily mounted in oil of cloves. The larger vessels were treated in a similar manner; and transverse sections of them, when placed beneath the microscope, showed the walls of many to be diseased. The lesion was limited almost exclusively to the adventitia and surrounding connective tissue. In general, only the outer part of the muscular tunic was affected. The

tunica intima showed scarcely any appreciable alteration. The lesion located in the outer coat of the vessel was characterised by a dense accumulation of embryonal cells in the meshes of its reticulum. Seen in longitudinal section, the vessels often presented a fusiform enlargement from point to point, an alteration which was restricted to the outer coat, and which rarely ever affected the calibre of the vessels. It was almost exclusively the arteries which were thus diseased.

"The sections of the cerebral cortex were made vertical to the surface of the softened convolution. They showed a variable number of caseous nodules, some of which were situated in the cortical substance near the surface, others in the thickened and inflamed pia mater. Upon minute examination, these caseous nodules were found to be composed of numbers of smaller caseous foci, massed together and united by an embryonal tissue, which presented the following peculiarities: in the portions at some distance from the minute caseous foci, embryonal cells were crowded together so thickly that it was impossible to discover any trace of the original structure. Scattered here and there in this embryonal mass were to be seen a number of what many authors figure and describe as 'giant cells.' On first view, and under a moderate magnification, these large multinucleated bodies certainly presented many of the characteristics of myéloplaxes; indeed, at the very edge of the minute caseous foci, where the embryonal tissue was on the border-land of caseous degeneration, it was altogether impossible from an examination of these bodies at this location alone to say that they were not genuine 'giant cells.' Recurring, however, to the more remote portions of the embryonal mass, where the cellular tissue was not nearly so much altered, by a very careful study, under a very high power given by an excellent lens, there was no difficulty in recognising peculiarities in these large multinuclear bodies, which demonstrated their nature and construction to be different from those of true 'giant cells.' In the loose altered spots of the embryonal mass could very readily be recognised the lumen, wall, and peri-vascular lymph-space of small arteriole and other blood-vessels. Many of these vessels presented an unobstructed lumen; and yet the walls of all of them were altered. The lesion of the patulous vessels was again mainly limited to the external coat, which, together with the outer layers of the middle coat, was represented by a mass of embryonal cells, constituting a cylinder. The endothelia of the tunica intima were swollen, and presented enlarged nuclei, but did not appear to be proliferating. Sometimes the peri-vascular lymph-space would be filled more or less completely with large

embryonal cells, but it was usually almost empty. The swollen vessels presented corresponding alterations. In some less altered spots, many of the vessels, in the same condition above described as to their walls, were plugged with a granular, fibrinous clot, which entirely filled the lumen, and which enclosed some white blood-corpuscles, a few detached swollen endothelia, and sometimes small numbers of visible red blood disks. In these cases a granular fibrinous coagulation more or less completely obstructed the perivascular lymph space also. In the intermediate region, between the above-mentioned altered portions of the embryonal mass and a narrow zone immediately surrounding the minute caseous foci, were many multinuclear bodies, in which the outlines of the vessels could still be faintly recognised by the arrangement of the nuclei. In this region were some also, in which, without the aid of a previous study, no suspicion of a similar origin and mode of formation could be suggested by their appearance, every semblance of a transverse cut of a vessel having been entirely lost; and yet, throughout the different portions of the embryonal mass, multinuclear bodies were so numerous, and of such varied appearance, that every gradation could be traced between the sections of undoubted blood-vessels, the external parts of which were in a state of proliferation, and the multinuclear bodies in the neighbourhood of a caseous focus, which gave no hint of such an origin.

"In the brain substance, and in the pia mater at the border of the embryonal masses which surrounded the caseous foci, the arterioles and the capillaries were altered in the following manner. Their external portions showed a cellular irritation throughout their entire length; at points the irritation had gone so far as to result in a nodular, or fusiform, or cylindrical thickening of the outer wall of the vessel. In the immediate vicinity of these altered vessels the tissue was in a state of inflammatory hyperplasia or inflammatory softening.

"From the foregoing study of the microscopical characters of the various lesions, it must be concluded that the specific morbid process in this case was that of tuberculosis.

"Longitudinal sections of one optic nerve, at its entrance into the eye, were made and prepared for study. Examination of these showed the nerve to be in the early stages of a descending optic neuritis."

This report speaks for itself, showing clearly the existence of tuberculosis, explaining satisfactorily the occurrence of the softening, and containing several points of special interest, such, for instance, as those relating to supposed "giant cells."

Remarks.—Although the autopsy revealed a somewhat diffuse meningeal inflammation, the only definite destructive lesions were those of the convolutions surrounding the fissure of Rolando. The inflammation, deposit, exudation and destruction of the cortex were greatest at the middle region of the left ascending convolution, where are situated the motor centres for the movements of the upper extremity. Motor paralysis of the right arm was the most marked of the paralytic manifestations. To explain the paralysis of the leg, we have thickening and adhesions of the pia mater over the paracentral lobule, with a small area of softening high up in the ascending parietal convolution. The nasal, oral, and lingual paresis have their explanation in the foci of softening and the tubercular granulations low down in the ascending convolutions. The lesions of the cortex were carefully and closely studied, a pencil-sketch, with measurements, indicating their intensity and exact location, being made at the time of autopsy, under my directions, by one of my resident physicians, Dr. Bell. Her disturbance of speech was an oro-lingual paresis rather than a true aphasia. The ptosis and late external strabismus, were probably due to the involvement of the oculo-motor nerve in the meningeal inflammation at the base. Both ophthalmoscopic examination and the microscope showed the association of optic neuritis with the meningitis.

CASE IV.—*Localised Tubercular Meningitis with Cortical Softening—Involvement of Oro-Lingual Centres—Internal Pachymeningitis of the Opposite Hemisphere.*

Y——, aged 65, was sent from the Blind Ward to the ward for nervous diseases, because he had had a slight apoplectiform seizure. I could only learn in regard to him that he had been weak and thin for a long time; that he had been troubled with cough and diarrhœa; and that he had complained frequently of headache and giddiness. His mind acted very feebly; he seemed to understand what was said to him, but he was somewhat obtuse and slow in answering. His speech was peculiar, it might be described as slightly "staggering:" a little tremor was present in the tongue and the muscles about the mouth; he sometimes dropped a letter or syllable. He was drowsy, dosing the larger part of his time. The only evidence of loss of power in the face, which was carefully studied, was about the mouth, which was drawn slightly to the left, and the right angle of which drooped slightly. His tongue did not deflect to either side, but its movements showed weakness and some want of control. The uvula and soft

palate were not paralysed. Neither ptosis nor strabismus was present. Cataract operations had been performed on both eyes, and he was nearly blind, having only quantitative vision. His limbs were all very weak, but no distinct motor paralysis, more marked in one than in the other, could be made out, except that the grip of the right hand was poorer than that of the left. His right leg, however, had been amputated a few inches below the knee, about five years before coming under observation, which prevented a full determination as to paralysis on this side. Physical examination of both lungs showed that wide-spread and advanced tuberculous disease was probably present. He was troubled with an obstinate diarrhœa, and died, in ten days after admission, of exhaustion, which was largely due to this diarrhœa.

An autopsy was held twelve hours after death. The skull was normal. The brain weighed forty-three ounces. The inner surface of the dura mater of the convex portion of the right hemisphere, from the middle of the parietal lobe forward to the anterior extremity of the brain, was covered with a layer, from half a line to a line in thickness, of a red, yellowish-red, and reddish-brown colour at different points. This stratum could be readily scraped from the dura, leaving this membrane intact beneath. The same formation, but of less thickness, extended down the right side of the falx. The pia mater, beneath the changed dura, presented a slightly opaque, bluish-white appearance. After stripping up this membrane, it was found that all the convolutions of the convex surface of the hemisphere, within the area covered by the diseased dura, were much flattened, and of a reddish-brown or light rusty colour.

The dura mater of the left hemisphere was unchanged. The pia mater of this side was hyperæmic, and here and there through it were seen peculiar-looking straight streaks of red. On removing the membrane from the substance of the brain it was adherent at several points, particularly along the fissure of Rolando. Miliary tubercles were made out at scattered points over the postero-frontal and parietal lobes.

On the surface of the left hemisphere were four foci of superficial softening, corresponding to points where the pia mater was more or less adherent to the cortex, and presented on its under-surface spots of yellowish exudation. The softening was only from one to two lines in depth, and was of a whitish-yellow colour; it had probably resulted from the obliteration of small vessels by the tuberculous process, which was more intense in the pia mater at these positions than elsewhere. These softened areas were situated as follows: (1) An

irregular area, five-eighths of an inch wide at its upper boundary, and narrowing below to one-fourth of an inch, at the level of the middle third of the fissure of Rolando, its posterior margin being eleven-sixteenths of an inch in front of this fissure; it included small portions of the second, third, and ascending frontal convolutions. (2) A spot, about the size of a half-dime, in the ascending parietal convolution, about the junction of the middle and lower thirds of the fissure of Rolando. (3) A spot, two-thirds of an inch vertically, and one-fifth of an inch wide, extending on both sides of and one-third of an inch below the lower extremity of the same fissure. (4) An area, five-eighths of an inch in width, and extending across the second temporal convolution, an inch backwards from the Sylvian fossa.

A very slight depression was found about the centre of the ventricular surface of the left corpus striatum.

Both lungs were infiltrated with miliary tubercles, the upper lobes being most affected. Cheesy metamorphosis had taken place in several localities, and an abscess had formed in the right apex. The cardiac valves were all-sufficient, and nothing abnormal was found in the examination of the heart except a single calcareous plate on the outer side of one of the aortic crescents. The right kidney contained a brown encysted stone, and two similar stones were found in the substance of the left kidney. Both small and large intestines presented a remarkable condition of ulceration; fifty-two distinct ulcerative foci being counted in the former, and twenty in the latter.

Microscopical examination confirmed the macroscopic evidences of the existence of tuberculosis of the pia mater.

Remarks.—In this case we have a tuberculosis of the pia mater of the convexity of the left hemisphere. The tubercles discovered were fewer in number, and the signs of inflammation were less marked than in the last case; speaking generally, the same portion of the convexity was attacked in both, but in the present case the base escaped. Limited tuberculosis of the pia mater is not so rare as many neurologists suppose.

In regard to localisation, it will be seen from a study of the areas of softened cortex that portions of the arm, face, and speech-centres were implicated. The softening was very superficial, and the paresis of the right upper extremity may or may not have been the result of the lesions; but I am inclined to attribute the peculiar speech and the paresis of mouth and tongue to the cortical destruction, as the oro-lingual centres of Ferrier were so distinctly involved. In regard to the softening of the second temporal convolution, I have nothing to say in

connection with the symptomatology of the case. The special senses could not be studied to any purpose.

The internal hæmorrhagic pachymeningitis of the right hemisphere was in itself a most interesting lesion, but a lengthy discussion of it is foreign to my purpose in the present paper. Although the case was one complicated by many conditions, some of the patient's symptoms were doubtless due to the cerebral compression, which the autopsy showed had been exerted by the chronic pachymeningeal disease. Drowsiness was one of these symptoms; and the headache, vertigo, and weakness of the left half of the body may also have been partly, at least, due to the dural affection. Pachymeningitis has not unfrequently been observed in cases of tuberculosis of the pia mater, lungs, and other parts of the body, and also in connection with atheroma and affections of the kidneys.

CASE V.—*Multiple Cerebral Embolism—Softening of the Corpus Striatum and Internal Capsule, and widely distributed Cortical Softening—Hemiplegia with Aphasia.*

C——, aged 45 years, when she came under my care, was not in a condition to give any information herself, and the few facts in regard to her previous history were obtained from her relatives and friends. During three years she had suffered with pains in her limbs. She was brought to the hospital on the 18th of July. Late in the preceding winter she had a stroke, which paralysed the right half of the body and affected her speech. Afterwards she never used more than three expressions, namely, "Yes," "No," and "Do you know?" She became irritable and emotional. On the 6th of July she had a second apoplectic seizure while lying down. She could not move from the position she was in, but was not unconscious. From this time she was helpless, was unable to feed herself, had involuntary passages, and had some fever with delirium at night. She often carried her left hand to the head, as if it was the seat of pain.

She appeared to understand much that was said to her when she was first seen, but she could not answer questions correctly. Her vocabulary now consisted only of the two words "Yes" and "No," and she used one of these for the other indifferently. The mouth showed a scarcely perceptible twist towards the left, so slight that I could not feel sure that it was not a deviation such as is sometimes seen in those in health. She could not get her tongue beyond her lips. She could swallow, but with some difficulty. She could open and

shut both eyes, and no noticeable paresis was present in the upper part of the face. The right upper extremity was powerless, and was wasted from the shoulder to the finger-ends; the fingers were slightly clawed. The right leg was also helpless, but not so absolutely as the arm; the limb was wasted, the thigh much more than the leg below the knee. She had no appetite; she did not seem to care for food at all. Sensation, roughly tested, seemed to be retained; she shrank from the pricking of the æsthesiometer, but this may have been partly a reflex manifestation. Patellar reflex on the right side was decidedly exaggerated, and a slight clonus could be developed from the right ankle. The above notes on her condition were made July 20th. She lived until the 30th, gradually growing worse, although she sometimes rallied a little for a few hours. She had more or less fever all the time, her night delirium continued, her pulse became frequent and irregular, and profuse sweating often occurred. Her temperature was taken both morning and evening, and in both axillæ, from July 24th to the 29th. The temperatures registered in the right axilla, the side paralysed, were higher than those in the left. The evening temperatures were usually considerably higher than those of the morning. The averages of the observations were as follows:—

	Right Axilla.	Left Axilla.
Morning	99·7° F.	99° F.
Evening	101°	100°·2.

A post-mortem examination was made twelve hours after death. The dura mater was paler than usual, and the pia mater was everywhere so anæmic and transparent that the convolutions could be readily traced before its removal. The brain weighed forty-one ounces. No less than seven areas of true cortical softening were found, as follows: (1) A spot, about two-thirds of an inch in diameter, on the left side, where the first frontal, second frontal, and ascending frontal convolutions come together. (2) One, a third of an inch in diameter, in the left third frontal convolution at the bottom of a secondary fissure. (3) A similar focus on the outer edge of the left island of Reil. (4) An irregular area, two-thirds of an inch in its greatest length, about the middle of the first temporal convolution of the right side. (5) A large foyer, which began an inch to the right of the longitudinal fissure, and just anterior to the transverse occipital fissure of Ecker, and extended backwards, expanding like a fan to the posterior limit of the upper surface of the right occipital lobe. (6) A similar foyer in the left occipital lobe, which began at a corresponding point, and spread in the same general direction, but only for about half

the distance of the softening on the right side. (7) A small spot, one-third of an inch in diameter, on the right posterior aspect of the cerebellum.

On cutting into the left lateral ventricle, a small yellow spot was visible on the surface of the corpus striatum at the beginning of its cue or tail. On incising this, the ganglion beneath was found to be softened and partly diffluent, the destruction being deep enough to involve a portion of the internal capsule. No other lesions of convolutions, tracts, or ganglia were discovered, after diligent search. The blood-vessels, particularly those of medium and small calibre, showed at intervals evidences of inflammatory changes in their walls. I succeeded in finding emboli lodged in vessels leading directly into the softened occipital areas. At a number of points in the branches of the middle and posterior cerebrals, at more on the left side, were seen what appeared to be plugs, or minute blood-clots, some loosely and some firmly lodged.

Pleuritic adhesions, which were particularly firm and dense on the posterior aspect of the left lung, were found on both sides. The lungs were œdematous, but contained neither deposits nor cavities. The heart was dilated, and its walls were in a condition of fatty degeneration. No valvular lesions, however, were present. Both kidneys were atrophied, granular, and tough.

The vessels were carefully stripped from the brain, and these, with specimens from the softened areas, were examined microscopically by Dr. E. O. Shakespeare, who reported as follows: "The walls of the vessels were in a state of cellular hyperplasia. The endothelial cells covering the trabeculæ of the finer fibrous network of the pia mater were swollen, granular, and sometimes contained more than one very distinct nucleus. In the meshes of the network in many places could be seen a coagulum of granular fibrine, enclosing white blood corpuscles, a few red ones, and some detached and swollen endothelia. In a few of the largest vessels an embolus could be distinctly recognised; and at the location of the plug, the inner, middle, and exterior coats of the vessels were much irritated and sometimes inflamed. The diseased cerebral substance presented at different points the usual appearances of inflammatory and necrotic softening. The pia mater attached to the softened cerebral tissue showed inflammatory thickening, and the appearances commonly present in subacute meningitis."

Remarks.—How shall we associate the symptoms presented by this case during life with the numerous lesions found after death? Such a multiplicity of diseased areas might at first

tend to throw us into confusion. The major portion of the dissociated hemiplegia was probably due to the softening of the left corpus striatum and adjacent internal capsule; the aphasia to this lesion, or conjointly to it, and to those of the third left frontal convolution and the island of Reil. The softening (1), at the junction of the first, second, and ascending frontal convolution, included the anterior upper portion of the general region for the superior extremity. It will be remembered that in the history given the paralysis and wasting were stated to be much more marked in the upper than in the lower limb. This fact was carefully determined, measurements of the limbs having been made both before and after death. The greater completeness of the brachial paralysis and wasting may have been attributable to the partial involvement of the general arm centre in one of the lesions, both the voluntary-motor centres in the cortex, and the sub-voluntary or automatic centres in the striate body probably being destroyed. The cortical areas of softening were not high enough to implicate the supposed centres for the leg; and the paralysis and atrophy of this member must, I think, be attributed alone to the lesion of the caudate nucleus and adjoining tracts. The wasting of the right thigh out of proportion to that below the knee, which strongly attracted my attention, is an observation not to be disregarded, although I have no special explanation to offer. I am satisfied that, with all the advances that have been made in this direction, we do not study our cases minutely enough. Many facts of value may yet be learned from the commonest examples of hemiplegia, by a closer study of symptomatology. Differences in the amount of wasting in various parts of the same limb, the greater preponderance of paralysis or spasm in certain muscles or muscular groups, and many other similar points, need to be more thoroughly investigated with reference to the effect and position of the lesions found post-mortem. The exact limitations, superficial and deep, of lesions of the basal ganglia, should be determined. In this case the head of the striate body escaped, but the softening extended below the caudate nucleus. Decided facial paralysis was absent, although a very slight paresis of the lower right facial region seemed to be present. The cortical facial zone, as usually given, just escaped. The tongue and the muscles concerned in swallowing were undoubtedly affected. Owing to the condition of the patient, hearing and the special senses generally could not be thoroughly investigated, so that I am not able to say whether any defect of hearing resulted from the temporo-sphenoidal lesion. In regard to the large foyers of softening in both occipital lobes, and the cerebellar

focus, if any inferences in regard to the lesions in other regions have been correct, the effects produced by these were latent, or were not of a positive character. Ferrier regards the occipital lobes as specially related to the visceral or organic sensibilities, but is doubtful whether I would be justified in referring such symptoms as impaired appetite and involuntary discharges, which occur in so many conditions, to any localised cerebral lesion. Sensation, so far as it could be tested, seemed to be unimpaired.

It may be worth while to give a passing notice to such symptoms as patella reflex, temperature, and fever and delirium. Very commonly, as in this case, in hemiplegics and monoplegics, tendon reflex is exaggerated on the paralysed side, owing probably, in part, at least, to the fact that the parts below the lesion are more or less cut off, or from the restraining influence of the higher brain. The temperature was slightly elevated, as in many instances of localised cerebral disease; it was also higher on the paralysed side than on the other. The case being one of demonstrable multiple embolism of vessels of appreciable size, Bastian's hypothesis in regard to fever and delirium being due to capillary occlusions becomes worthy of consideration. The conditions were such as to bring about such occlusions.

Pathologically, this case is a most interesting illustration of multiple cerebral embolism. It is probable that on several occasions particles from a partially-organised blood-clot were carried into the circulation, and lodged in the cerebral vessels. Some of these became permanent fixtures at their points of lodgment; others were immediately swept out again. Necrotic softening took place in some instances in the districts supplied by the occluded vessels; in others this process was probably prevented by collateral circulation. As has been noted, plugs were discovered in a number of places in branches of the middle and posterior cerebral arteries, these in some cases apparently not having caused necrobiosis. In some recent experiments made in conjunction with Dr. A. J. Parker, of Philadelphia, we found the communications between the three chief vascular territories of the brain freer even than is held by Heubner, and far freer than Duret would have us believe. Our inspections so far have been made only on two brains, and great individual differences may and probably do exist; but the fact that in these two sets of experiments we found communicating vessels at least two millimetres in diameter, shows that parts of the brain may frequently be saved from softening by anastomosing vessels. This is contrary to the views advanced by me in a Review of the subject of *Cerebral Localisation*

('American Journal of the Medical Sciences,' July 1879), in which I state that up to the time of writing, my clinico-pathological observations were corroborative of the views of Duret, Cohnheim, and Charcot. Subsequent pathological experience, and the experiments just referred to, have, however, led me to a somewhat different conclusion, although I do not yet feel satisfied in regard to the question. Multiple embolism is probably more frequent than is usually supposed, occluded vessels which do *not* occasion softening often escaping attention.

The inflammation of the pia mater was confined to that portion of the membrane in immediate relation with the necrotic districts.

The inflammation of the walls of the vessels, which was so marked, may have been partly aroused by the irritation produced by the numerous embolic foci, partly it was due to these vessels coming from the zone of inflammation around the softened areas.

The co-existence of fatty degeneration and dilatation of the heart with contracted kidneys affords a plausible explanation of the formation of the supposed clot. A tendency to the production of cardiac and other thrombi is well known to exist in diseases such as fatty heart, which cause marked impairment of the power of this organ.

The areas of softening observed were more numerous in the left than on the right half of the brain, which is in accordance with the well-established and easily explained fact that emboli are much more frequently carried into the left carotid than into the right. The occurrence of bilateral softening of the occipital lobes is deserving of passing attention. A tendency to bilateral embolism has been noted by several observers.

CASES OF INTRACRANIAL TUMOUR.

BY JAMES ROSS, M.D., MANCHESTER.

CASE 1.—JOHN THOMAS GOULD, æt. 14 years, was admitted into the Royal Infirmary on March 5, 1877, under the care of Dr. W. Roberts, to whose kindness I am indebted for permission to publish the case. He was a strong and healthy boy until a few months ago; his parents were also healthy, and there was no family history of consumption or of any other constitutional disease. He was a brick-setter by trade, and three months previous to admission fell from a ladder and struck the back of his head on the pavement, and since that time he has suffered from more or less constant occipital headache.

Condition on admission.—As he lies in bed he can move his legs freely in any direction; but on attempting to walk, the feet are alternately projected forwards, and the heel comes down with a thump, producing a gait similar to that of locomotor ataxy. He cannot maintain the erect posture unsupported; and when all external aid is momentarily withdrawn, his head shoots downwards and forwards, as if the body were about to revolve round a transverse horizontal axis. When the patient is caught in the act of falling and raised again to the erect posture, he complains of dizziness, and sees objects revolving from right to left. On directing his eyes to the right, a slight oscillatory movement of the eyeballs is observed, but there is no nystagmus when he looks straight before him. The patient is almost quite blind in the left eye; but he can distinguish objects clearly with the right eye.

When the right eye is fixed on an object, such as a finger twelve inches in front, a second finger moved laterally and to the right is seen until it is almost nine inches from the first, showing that the field of vision to the right is not sensibly diminished. But starting again from the finger on which the eye is fixed, and moving the second laterally to the left, the

latter disappears from view when it is from one to two inches removed from the former. I say from one to two inches, because his replies are not always the same, thus indicating that the sensitive and blind portions of the retina are not separated from one another by a sharp and defined border, but fade insensibly into one another. The pupils are dilated, equal, and respond slowly to the stimulus of light. An ophthalmoscopic examination reveals double optic neuritis with swollen disc; but there are no patches of exudation or atrophy. The other special senses and the mental faculties are unaffected, the urine is free from sugar and albumen, the appetite is good, and all the other functions of life are normal. He was ordered iod. potass., grs. x., to be taken three times a-day.

March 31st, four weeks after admission.—It is evident that the symptoms have altered considerably. The patient now habitually lies either on his back, or slightly inclined to one side. He cannot sit erect without support, but he has still some degree of voluntary control over his legs, although the movements are feeble. Cutaneous sensibility is impaired in the lower extremities; he can still feel when touched, but he cannot localise the touch well. The sensibility to variations of temperature and to pain is also impaired. There is complete blindness of both eyes, but there is only slight atrophy of the optic discs. Two days ago he passed his urine in bed for the first time. His bowels are very constipated, and there is general emaciation.

April 15th.—He complains much of frontal headache. There is complete anæsthesia, and entire loss of voluntary motion of the lower extremities; the stools and urine pass involuntarily, and there is a large bed-sore over the sacrum. He cannot hear the ticking of a watch so well with the left as with the right ear.

May 14th.—There is complete anæsthesia of all parts below a line passing round the body on a level with the anterior superior processes of the ilium. Reflex irritability is entirely abolished in the lower extremities, as tested by tickling, pricking, and the faradic current. The muscles of the legs and thighs do not respond in the slightest to the faradic current. The calf of the right leg measures $9\frac{1}{4}$ and that of the left only $7\frac{1}{4}$ inches. Each thigh measures $9\frac{3}{4}$ inches. The muscles of the thighs and of the right leg appear only to be emaciated in proportion to the rest of the body; but it is manifest that the muscles of the left leg are specially atrophied. There are deep bed-sores over the sacrum, the prominences of both thighs, the external malleoli of both

ankles and the inside of the left knee. The intelligence is greatly blunted, and he lies in a half stupor, but replies readily to any simple question asked him. His pulse is very feeble, and beats about 140 in a minute; but he still eats any food which the nurse brings him. About a week ago it was noticed that the left eyelid was only half closed when he was asleep, and he could not close it entirely by a voluntary effort. There was also slight paralysis of the left facial muscles, so slight that the difference between the two sides could scarcely be detected when the face was quiescent, but was recognisable when the patient smiled. This affection of the facial nerve only lasted a few days, and has now entirely disappeared. The state of his hearing cannot be satisfactorily tested, owing to his apathetic condition.

From this time he lingered on without any further special symptom manifesting itself. He became more and more apathetic and extremely emaciated, and died on July 3rd, four months after entering the Infirmary, and seven months after the fall, to which in all probability the origin of the disease is to be traced.

Sectio cadaveris, twenty-four hours after death.—The body is greatly emaciated. The calf of each leg measures 7 inches, and each thigh 7¼ inches. The sacrum, both trochanters, and the external malleolus of the left foot are exposed and denuded in consequence of extensive bed-sores. The tips of both ears are also ulcerated, as well as the inside of the left knee.

On removing the calvarium the brain appears to project, and the convolutions are flattened. The sinuses and the veins on the surface of the brain are gorged with blood. The substance of the brain is somewhat soft, and the ventricles are distended with fluid; but the cerebrum is in other respects healthy. Some grumous turbid fluid escaped from between the cerebellum and corpora quadrigemina during removal of the brain. On inspecting the cerebellum the edge of a tumour is noticed between the anterior surface of the middle lobe at its inferior margin and the posterior surface of the medulla oblongata; while the superior end of the tumour can be seen between the cerebellum and corpora quadrigemina. On making a vertical section of the cerebellum in the middle line down to the floor of the fourth ventricle, the tumour is seen to occupy the whole of the anterior part of the middle lobe of the cerebellum, being somewhat more developed on the right than on the left side. In consistence the tumour is soft, brain-like, of a greyish-red colour, the centre being so softened as to have left a small cavity from which the turbid fluid already mentioned had escaped. The tumour is not circumscribed;

its margins gradually blending with the surrounding nervous tissue. The growth passes along the right superior peduncle of the cerebellum to reach the corpora quadrigemina, and the latter are somewhat softened and flattened. On opening the spinal canal the cord is seen to occupy the whole of the cavity transversely, the diameter of the cord being about one-third larger than that of a healthy cord. The whole of the cord feels brawny, like bacon. On making transverse sections from above downwards, half an inch apart, the membranes (the cortical layer of the neuroglia?) in the cervical region are seen to be slightly thickened, and the cord is softened; but in the upper dorsal region the spinal pia mater, especially on the posterior aspect, is three times as thick as in a healthy cord. In the middle of the dorsal region the new growth forms a dense well-defined tumour, which pressed on the cord from behind forwards, so that only a small part of the anterior segment of the cord is left. In the lumbar region, again, the tumour surrounds the cord, so that a central core, about the size of a goose quill, of softened nervous tissue is all that is left to represent the spinal cord. The substance of the tumour is of the colour and texture of bacon, and much denser than the tumour of the cerebellum.

The left lung is adherent to the chest walls. Both lungs are healthy. The heart is normal. The abdominal organs are healthy. Subsequent microscopic examination showed that the tumour consisted of small delicate cells imbedded in a finely granular substance. The growth in the cord presented similar microscopic characters to the cerebellar tumour; but contained a larger amount of intercellular substance.

Remarks.—The first time I examined the patient, my diagnosis was "tumour, probably a glioma, situated in the inferior portion of the middle lobe of the cerebellum, and pressing forwards on the corpora quadrigemina." The reasons for regarding the case as one of intracranial tumour are so manifest as scarcely to require mention. They are: the history of an injury to the head, the gradual development and progressive character of the symptoms, the constant headache, and the existence of double optic neuritis. My reason for believing that the tumour was situated in the inferior portion of the middle lobe of the cerebellum was the remarkable manner in which the head and shoulders shot forwards and downwards, as if the patient were about to revolve round a horizontal axis. The circumstances in favour of the tumour being a glioma were these: if the diagnosis with respect to the localisation was correct, the tumour probably grew in the substance of

the nervous tissue itself, and not from the membranes; the appearance of the patient and the family history were against tubercle; there was no evidence of congenital syphilis; and the age of the patient put cancer almost out of the question.

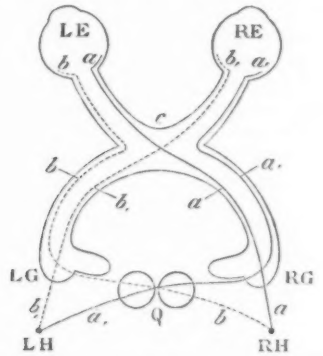
My reasons for thinking that the tumour pressed forwards upon the corpora quadrigemina were that I thought this would to some extent explain the excessive staggering present during attempts at walking; but much more that it would explain the great impairment of vision present at such an early stage of the disease. But the peculiar character of the disorder of vision demands an explanation; and for this purpose let us take Charcot's scheme of the decussation of the optic tracts as our guide.

In this case, vision was almost totally lost in the left eye, and the state of vision of the right eye simulated nasal hemiopia. I say simulated, because it was not a case of hemiopia at all. The condition of vision in both eyes was that of amblyopia in its progress towards amaurosis. The amaurotic condition was nearly reached in both sides of the retina in the left eye; but in the right eye the right half of the retina had become amaurotic, while vision was tolerably good in the left half; and between the two halves of the right retina there was a relatively broad border land, where the comparatively good vision of one side faded gradually into the blindness of the other. Such a condition as this could not, therefore, have been caused by the pressure of a tumour on the commissure or optic tracts; it was not likely to have arisen directly from the optic neuritis, inasmuch as there was no atrophy of, or patches of exudation on the disc; it must therefore have been caused by a lesion interfering with the optic fibres, either directly or indirectly, beyond the termination of the optic tracts in the external geniculate bodies.

It appeared to me very probable that a tumour pressing on the right side of the corpora quadrigemina and extending gradually to the left, would produce the condition of vision met with in this case. The whirling of objects from right to left which the patient described, showed that there was a greater amount of irritation of the right than of the left lobe of the cerebellum, and this rendered it probable that the tumour of the middle lobe extended farther to the right than to the left.

Suppose, then, that a tumour is pressing on the corpora quadrigemina from behind forwards, and from right to left, the fibres ($b\,a$) coming from the left eye, and meeting at R H, would be first interfered with, then the fibres (a') coming from the right half of the retina of the right eye, would be intercepted

in their passage behind the corpora geniculata to the opposite side. The fibres *b'* coming from the left half of the retina of the right eye would be the last to be injured, so that the condition of vision which was present in this case would be produced. It may be urged that Charcot's scheme of decussation is merely diagrammatic, and that the points L H and R H are supposed to represent positions in the cortex of the hemispheres; but my reply is that I am only making a diagrammatic use of it. If there is a semi-decussation of the optic nerves in the chiasma, and if the fibres which do not cross in that place decussate behind the corpora geniculata, then, whatever may be the further course of these fibres, some such effect as that



L E, and R E, left and right eyes; C, chiasma; L G, and R G, left and right geniculate bodies; Q, corpora quadrigemina; L H, and R H, left and right centres of vision: *b* and *a* nerve-fibres from left and right sides respectively of left eye; b^1 and a^1, corresponding fibres from right eye.

indicated would be produced by a tumour pressing from behind forwards, and from right to left on the corpora quadrigemina. This at least was the process of reasoning by which I came to the conclusion that the case was one of tumour of the anterior part of the middle lobe of the cerebellum, inclining to the right side, and pressing forwards on the corpora quadrigemina, and this conclusion was verified to a considerable extent by the post-mortem. One serious objection I always felt to this view was, that it was not manifest how the floor of the fourth ventricle could escape under such circumstances; and yet there was no sugar in the urine, no polyuria, and the breathing was uninterfered with. The autopsy explained this. It showed that the corpora quadrigemina were probably not so much interfered with by pressure as by extension of inflammation

and subsequent softening, and probably also extension of the glioma along the superior peduncle of the cerebellum into the substance of the corpora quadrigemina.

As the case progressed, it soon became evident that there was an independent affection of the cord, as evinced by the complete anæsthesia, and loss of reflex irritability in the lower extremities, as well as by the trophic changes already described.

Two suppositions could be made with regard to the affection of the cord. Either there was a tumour pressing on the cord of the same nature as that in the cerebellum, or there was extensive softening of the lumbar region. I must acknowledge that although the latter view was not free from difficulties, it was the one I adopted. In reference to the first supposition, I was only thinking of a circumscribed tumour pressing on a limited part of the cord, and this would not account for the early and total loss of reflex irritability in the lower extremities. It never entered into my calculations to think of a tumour extending the whole length of the cord. Looking back on the case with the further light which the post-mortem has thrown upon it, it seems probable that the functions of the posterior columns of the inferior dorsal and lumbar regions of the cord were interfered with by the growth of the tumour, even as early as the date of admission. This would account for the projecting forwards of the legs, and thumping of the heels, which were so like the movements of locomotor-ataxy.

(To be continued.)

A CASE OF HEMIPLEGIA DUE TO WASTING OF CEREBRAL CONVOLUTIONS.

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and

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THIS case presented the following congenital defects, Bell's paralysis associated with disease and deformity of the right ear; congenital cyanosis, and left hemiplegia associated with destruction of certain convolutions in the right hemisphere.

The patient, Emma B——, was a girl aged 1 year 9 months, and came under observation at the East London Hospital for Children as an in-patient, July 1878. She was ill-nourished and ill-developed, exhausted, and very cyanotic, manifestly very deficient in general muscular strength, being unable to stand or sit up. She generally lay on her back, quite passive, but appeared to know her mother, and would look about her, turning her head towards any object that attracted her attention; she had not learnt to speak, but cried like other babies, and made her wants known. With the right hand she would hold and play with a toy, moving the fingers well; the voluntary movements of the right lower extremity also appeared complete. The left arm and leg remained almost constantly inactive, with only occasional very slight movement, and that purposeless and apparently quite involuntary.

On slightly pricking the left arm or leg, it was drawn up; if more strongly irritated, she tried to remove the offending pin with the right hand, but was quite unable to use the left limbs for any voluntary purpose. The left limbs were easily excited to reflex contraction, and muscular contraction to the faradic current was well marked; cutaneous sensibility appeared unimpaired. Measurement indicated no difference

in the length of the corresponding limbs; there was no difference in their circumference on the two sides, and no rigidity. The skull was not deformed, but the right external ear was very rudimentary, smaller than the left, ill-shapen, and adherent to the skull, the tragus being the only part fairly well formed; the meatus was large in its vertical diameter, and there was a depression above the meatus running inwards to a blind termination ¼-inch from the surface. There was much purulent discharge from the ear.[1] The left ear was fairly well formed, but was also the seat of otorrhœa.

The neck was very short, and its movements were limited laterally, but the head could be rotated to the due extent by passive movement. There was complete Bell's paralysis on the right side, which had existed from birth. On blowing in the child's face she winked with both eyes, but closed the right eyelids less completely. There was no paralysis of any other cranial nerve; she was readily attracted by sounds, sight was manifestly present, and the ophthalmoscope showed no abnormality; there was no difficulty in taking liquid food.

The chest was fairly well formed, and the lungs appeared healthy. The cardiac impulse was forcible, suggesting some ventricular hypertrophy; the apex beat was displaced slightly outwards; the area of cardiac dulness was well-defined, but was not perceptibly enlarged. A loud systolic bellows murmur was heard all over the precordial region, but with greatest intensity over the second left cartilage. The pulse was full and strong, cyanosis was very pronounced and constant, the fingers and toes were slightly but distinctly clubbed.

The following history was obtained from the mother. The child was born at full term. The mother while pregnant had a fright about the fourth month, but was not aware of any ill effects; at the sixth month she had a fall. The parents are both intelligent, strong, and of steady habits, and have had eleven children, all alive and healthy. At birth the deformity of the ear and neck and the facial palsy were at once noticed. Cyanosis is said to have been more marked during the first fortnight of life than later. The child could never suck, and was fed from a bottle. The weakness of the left limbs was not noted till a later period, but it appears certain that it must have existed from birth. When nine months old she was convulsed for thirty-six hours; it is said the right arm alone worked, the left remaining quiet. After this she was very exhausted for a week, and then rallied; but from this time the left arm was quite useless. No other convulsions occurred.

[1] For this sketch we are indebted to Mr. Basil Walker, late House Physician at the London Hospital.

The child was subsequently received into the Darenth Asylum; no special symptoms occurred, and she died of bronchitis, Feb. 23rd, 1879.

Necropsy sixty hours after death. Body emaciated. Skull symmetrical. Calvaria thin, and with difficulty separated from the dura mater.

Brain weighed 31 ounces. The right hemisphere was smaller than the left, and after preservation in spirit for about three months, weighed 4¼ ounces, and measured 11·5 × 9 decimetres longitudinally. The pia mater was healthy at the summit of the hemisphere, slightly thickened over the frontal and occipital lobes, more thickened and absolutely diseased over an area measuring 5 × 4·25 decimetres. The convolutions included in this area were the lower part of the ascending frontal and parietal, the whole of the supra-marginal, with its lobule and the superior temporo-sphenoidal. The pia mater in this region was thick and tough, its vessels very large, tortuous, and contained blood.

Diseased convolutions.—The ascending frontal, at a point 4·5 deci. from its summit, suddenly became shrunken to the thinness of a wafer; the nerve matter was then continued in its usual direction downwards, partially forming the roof of a large adventitious cavity in the substance of the hemisphere.

The ascending parietal convolution, at a point 6·0 deci. from its summit, suddenly underwent a similar change.

The whole of the supra-marginal, with its lobule, was shrunken and almost destroyed.

The superior temporo-sphenoidal was much wasted. On examining it, a cavity was found lying between the layers of the wasted convolution, and lined with fibrous tissue.

The adventitious cavity lying below the wasted convolutions (except the superior temporo-sphenoidal) measured 5 × 3 deci.; and was also lined with thickened fibrous tissue, a band of which passed across it, forming a septum. The floor, generally smooth, with the exception of a projection the size of a hazel-nut situated at its posterior part, was composed of a very thin layer of nerve tissue forming the roof of the lateral ventricle. Neither of these cavities appeared to be a serous or blood cyst, or the sac of an abscess. They did not communicate with one another.

The lateral ventricle appeared normal; its ganglia and the choroid plexus were healthy.

The rest of the brain was healthy, and the convolutions were of normal size.

The head only was allowed to be examined.

Case of Hemiplegia due to Wasting of Cerebral Convolutions. (Warner & Beach)

Remarks.—In this case it appears certain that the left hemiplegia existed at birth, and was due to intra-uterine wasting of the convolutions described, for during life there was no illness (except one attack of convulsions) which could account for the large amount of localised cerebral atrophy. The hemiplegia was characterised by total loss of voluntary power; but sensation, reflex action, electric contractility, and good nutrition remained. There was no spastic contraction or convulsion of the affected side. It may be noted that the seat of atrophy included a considerable area of Ferrier's motor regions. The congenital ear defect was obviously connected with Bell's paralysis, and it is noteworthy that it existed on the same side as the cortical lesion. The congenital cyanosis appears to lend force to the argument that the disease of the brain was of intra-uterine character.

Abstracts of British and Foreign Journals.

On the Sensory Centres in the Cortex Cerebri. Luciani and Tamburini (*Studi Clinici sui Centri Sensori Corticali, Comunicazione preventiva dei Professori Luigi Luciani e Augusto Tamburini. Milan*, 1879).—These distinguished observers have endeavoured to collect cases where lesions in the brain were found in connection with deficiency of any of the senses, in the hope of throwing light upon the new localisations of sensory centres in the brain, attempted by Munk, Ferrier, and others. They commence with the announcement that Schiff has at length been induced by the clinical researches of Charcot and Pitres, and those of Maragliano, to admit the existence of such centres in man. From different authors they have collected forty-one cases of lesion connected with sensory deficiencies, thirty-four of which are failures of vision, (amblyopia, hemiopia, blindness, &c.). In three there was deficiency both of sight and hearing; in one the hearing alone was affected, and in three more the lesions were of a diversified character. The authors find themselves led by their inquiries to admit that the psycho-sensory centres of vision are situated both in the angular gyrus and in the occipital lobe. In this way they at once reconcile the rival theories of Ferrier and Munk, and get rid of negative observations. The cases collected are taken from medical writers in several European languages. Many of them, not having been composed with a view to illustrate these novel theories, are wanting in detail.

Out of thirty-four cases of deficiency of vision, the pathological lesion was in thirteen cases in the right hemisphere; in thirteen in the left; and in eight in both hemispheres. Of the thirteen instances in which the lesion was in the right hemisphere, the deficiency of vision was in five cases in the left eye; and in eight it appeared to be in both eyes. In the cases in which the lesion occupied the left side, four had deficiency in the right eye, and in nine the deficiency was on both sides, or, at least, the

loss of sight was not limited to one eye. In fourteen cases the lesion occupied one spot alone; in eight of these it was in the parietal lobe, in two of the eight it was in the angular gyrus. In six cases, it was in the occipital lobe.

In two cases of deafness there were lesions of the temporal convolutions.

The authors consider the results of their inquiry to be at least promising.

They present the following conclusions:—

1. Clinical observations confirm the results obtained in our experiments on animals, as to the localisation of the sensory zone in general and of the visual centre in particular.

2. The negative clinical cases may be regarded either as inquiries imperfectly conducted or recorded, or as lesions where the whole sensory zone was not implicated. Or perhaps the slowness with which the disease advanced allowed time for compensation, either by the function being vicariously assumed by the centre on the opposite side, or by the nasal secondary centres.

W. W. IRELAND.

On a Case of Progressive Muscular Atrophy, with Lesions in the Anterior Horn of the Cord. ERB and SCHULTZE (*Arch. f. Psych. u. Nerv. Kr.* ix. 369).—The publication of this case is intended as an answer to that of Lichtheim (ibid. viii. 1878), which the latter gave as one of *typical* progressive muscular atrophy, without lesion of the cord. Strong doubts are raised by Erb and Schultze as to the correctness of the diagnosis: first, because the clinical evidence is not in concordance with it; second, because the post-mortem appearances of the muscles showed them to be affected with simple, not with *degenerative* atrophy. The former is characterised by a mere quantitative diminution of all the constituents of muscular tissue; the latter, by proliferation of nuclei, loss of striation, waxy degeneration, &c.

In the present case the clinical characters corresponded with the classical picture of the disease. The post-mortem appearances of the muscles were both to the naked eye and to the microscope typical. Macroscopically the cord presented no change. Hardened and stained sections showed changes in the central part of the grey matter. There almost nothing but Deiter's cells, some of them much enlarged, are to be seen. The ganglionic cells of the middle group are much degenerated; those of the anterior and postero-lateral groups almost all normal. Clarke's columns and all the

white matter normal. Anterior roots normal throughout. There was then no continuity between the seat of spinal and of muscular lesions, a fact which gives support to the theory that there are trophic centres for the muscles independent of those of the nerves. Now the question arises: Which is here the secondary lesion? Did the nerve-centres degenerate in consequence of the muscular atrophy, or *vice versâ?* Against the former view the authors adduce a number of arguments; and without wishing to appear dogmatic, conclude that the muscular depends upon the spinal lesion.

On Concussion of the Spinal Cord. OBERSTEINER (*Medizinische Jahrbücher*, iii. iv. 1879, and *Separat-Abdruck*, pp. 32).—By this expression the author understands those cases where the spinal functions are altered in consequence of mechanical violence, direct or indirect, to the vertebral column, there being no anatomical lesion of the cord to account for such alterations. There is a "chronic" form of concussion, hitherto little studied, due to a series of minute shocks, which the author has observed among those employed on the tramways, and which manifests itself as weakness and unsteadiness of the legs. The ordinary form of concussion may be due to (1) falls or blows, (2) gunshot injuries, (3) railway accidents.

The author describes the case of a soldier who was shot in the nape of the neck, the bullet lodging near the third dorsal vertebra, without injuring the bony canal. He immediately felt intense pain in the legs, but could not move them. Three weeks after he had complete paraplegia and anæsthesia as high as the umbilicus, paralysis of the bladder with incontinence of fæces, extensive decubitus, &c. A careful post-mortem examination was made, which revealed extensive peritonitis and inflammation of the urinary organs. The middle third of the dorsal cord was diffluent, and the meninges there thickened and inflamed. Above and below, the large cells of the anterior grey matter presented alterations in the shape of vacuoles, or colloid drops in the protoplasmic constituents, alteration in the shape of the nucleus. Other cells were studded with numerous particles, which readily took the carmine stain. The other constituents of the cord, and the nervous roots, also showed degenerative changes. Below the seat of the main lesion the lateral columns were degenerated, and more or less extensive softened portions of the cord, with loss of substance, were noticed. Above, the posterior columns, together with the posterior

peripheral portion of the lateral columns, and Flechsig's "lateral fasciculi from the cerebellum" showed signs of ascending degeneration, which could be followed, in the case of the columns of Goll, into the medulla, in the case of the lateral columns into the corpora restiformia.

Discussing the symptoms of concussion, the author points out that it is necessary to distinguish between general and partial concussion; and also between the cases where the effects are immediately felt, and those where the symptoms are insidiously developed in time, as happened in 43 per cent. of the cases he tabulates. The prognosis is far more serious in the latter.

The leading symptoms are paralysis and anæsthesia of the extremities, and paralysis of the bladder and rectum, to which numerous others associate themselves differently in different cases.

As a rule, the pathological process, apart from secondary degenerations, is diffuse, and not limited to any system of fibres or cells. Tumours occasionally owe their origin to concussion.

The phenomena of concussion have been sometimes ascribed to vaso-motor effects; but the author refutes this theory, and believes that they are due to molecular changes in the nervous elements of the cord, whose immediate effects may vary, and which subsequently may undergo regression, or lead on to myelitic processes. Obersteiner brings his paper to a close with some remarks on secondary degenerations generally, showing the importance of the view that all the fibres undergoing ascending degeneration are centripetal to the cerebellum only. With reference to their pathogeny, he says that the blood-vessels are not primarily implicated. Nor does he agree with Erb in the viewing these secondary degenerations as "trophic" changes. He believes they originate in the lymphatic spaces which convey, upwards or downwards, the products of the myelitic softening at the point of lesion.

On Localisation of Atrophic Spinal Paralyses. Remak (*Archiv für Psychiatrie*, ix. 3. *Separat-Abdruck*, pp. 126).—In a previous paper (ibid, vi. 1) the author had discussed the probable spinal origin of lead palsy, and pointed out the great similarity between it in its generalised form and the anterior poliomyelitis of the adult. He had also shown that the exemption of the supinator longus in saturnine wrist-drop also occurred in non-toxic forms of spinal disease. Other muscles, such as the tibialis anticus and sartorius, are frequently found to remain intact, whilst all the other muscles supplied by the same nerve undergo atrophy. It is

to be remarked that electro-diagnosis gives us no direct clue to the spinal localisation of the original disease, but simply to the distribution of the atrophic process among the muscles; the former is to be inferred by reasoning from the latter. Again, it is evident that disease of the anterior roots will present the same clinical picture, as disease of the spinal centre of these roots themselves. Such is the case, for instance, in lesions of the fifth and sixth roots of the cervical plexus.

From a purely physiological point of view it is probable that various simple forms of movements depending upon co-ordinated action of several muscles, depend upon centralised mechanisms in the cord. But it is upon clinical evidence that we must mainly rely for the present in localising disease of the anterior horn of grey matter.

In the present paper Remak adduces numerous observations of his own, which he fully describes and minutely discusses, and shows how, so far, the results of autopsies concord with his results. Of these, we must content ourselves here to give but a bare outline. With reference to the spinal paralyses of the arm, he distinguishes an upper arm and a forearm type.

In the first the deltoid, biceps, brachialis anticus, and supinator longus, are affected together. When one of these muscles is seriously impaired, traces at least of nutritive changes are always to be discovered in the others by electrical investigation. But they frequently are visibly affected all at once (e.g. in cases of poliomyelitis, infantile or adult); and a case of lead palsy is adduced, in which this was the case. This state of things curiously coincides with that observed in some cases of lesion of the brachial plexus (Erb, Remak, Hoedemaker); and it must be remembered that the muscles just mentioned are those made to contract by exciting the fifth and sixth roots of the cervical plexus, at the supraclavicular point. Their spinal centre is situated high up in the cervical enlargement; and this explains the immunity of the supinator among the forearm-muscles, supplied by the musculo-spiral in cases of lead palsy and the like.

In the well-known forearm type, the muscles implicated are the extensors of the fingers and wrist. Their spinal centre is probably in the middle of the cervical enlargement. The extensor ossis metacarpi pollicis, seems to be centrally connected more with the inter-ossei, short-thumb, and flexor muscles of the hand. Their centre would be in the lower portion of the enlargement; and this assumption is supported by direct anatomical proof.

What precedes is based mainly upon observation in atrophic paralyses. The distribution of progressive muscular atrophy does certainly not present such clear types; but in many instances is in sufficiently close conformity to them, to indicate the spinal pathogeny of the disease.

With reference to the thigh, in the domain of the crural nerve, we find the sartorius enjoy considerable immunity. On the other hand, Remak mentions a case where it was found implicated along with the ilio-psoas. [This fact, taken in connection with the anatomical configuration of the lumbar plexus, seems to justify the assumption that the flexors of the thigh, of which the sartorius is one, are supplied by the second lumbar nerve.] The nucleus of the tibialis anticus is near that of the vasti and rectus femoris, with which it is often found to suffer. At the same time it must enjoy a certain independence, since this muscle is frequently found alone implicated; and, on the other hand, it is rarely affected along with the peronei and extensor muscles of the toes. [This would point to the fourth lumbar, as containing the fibres to the tibialis anticus.] In lead paralysis of the leg it has never been found paralysed, thus resembling the supinator in the arm. Autopsies point out the mid-lumbar enlargement as containing its motor nucleus.

The muscles of the shoulder-girdle and pelvis do not yet admit of spinal localisation. No leg types can yet be framed, as paralysis, though most frequently implicating the extensors, not infrequently extends to the flexors in various combinations.

Lesions of plexus roots imitate the distribution of poliomyelitic disease, from which other circumstances, such as sensory troubles, must help to distinguish them.

A. DE WATTEVILLE.

On the Structure of the Cortex Cerebri. STRICKER and UNGER. At the meeting of the Acad. der Wissensch. of Vienna, on July 10, 1879, Prof. Stricker and Dr. L. Unger brought forward the results of a research on the structure of the cortex. The following are the main conclusions:—

(1) In the cortex there is a single fine network, composed of fibres both of nervous and connective tissue.

(2) This network is composed partly of the processes of the ganglion cells, or their axial prolongations, and partly of the processes of connective tissue-corpuscles.

(3) Every variety of transitional form between connective tissue-corpuscles and proper nerve cells is found.

(4) The meshes of the network are filled with a fine granular or homogeneous substance.

(5) This network, with its substance, is analogous to the ground-substance of bone, cornea, and other connective tissues.

Shrivelling and Atrophy of the Cornu Ammonis in Epilepsy.—Dr. L. Pfleger, of Vienna (*Allg. Zeitsch. f. Psychiatrie*, Bd. 36), has had opportunities of post-mortem examination in a large number of epileptics—43 in all—and finds that in somewhat over the half of the cases, one or both cornua are shrivelled or atrophied; the unilateral degeneration being once again more common than bilateral. In his cases the degeneration was especially common in women, but this difference does not exist when all the recorded cases are taken together. From his table it also appears that in unilateral shrivelling the left is more commonly affected in males; whereas in females it is the right; in both cases in the proportion of 7 : 15.

A Case of Cortical Epilepsy.—Prof. Drasche (*Wien. Med. Wochensch.* No. 39, 1879) records the following case. A tubercular patient, aged 24, observed on July 14 a powerful contracture in his left wrist, and rigidity of the fingers. This passed off, but recurred on attempts at using the hand. This was soon followed by spasms of the fingers of the left hand. On the 18th the left side of the face, and both limbs (especially the upper) on the left side became paretic. Slight sopor. On the 19th instant epileptiform convulsion, with loss of consciousness; the convulsions affecting the left side. This was followed by complete left hemiplegia. On the 21st two similar attacks. Coma became deeper, and death on July 28.

Section.—Old standing tubercle of lungs. Chronic tubercular meningitis on the convexity of right hemisphere. On the anterior and posterior central convolutions (ascending frontal and parietal), several tubercles of the size of peas, invading the cortex, and surrounded by softening. Acute basilar meningitis.

H. Obersteiner.

On the Pathological Anatomy of Hydrophobia. Weller (*Arch. f. Psych.* ix. p. 493).—The following conclusions are founded upon an examination of the nervous systems of seven dogs and one man, death in all cases having resulted from hydrophobia. (1) Hydrophobia shows itself in the nerve-centres as an inflammatory process, originating in the vessels. It is characterised by

perivascular exudation, and infiltration of the tissues with lymphoid elements; the leucocytes are aggregated, more or less closely, in clusters or foci. At the same time, and probably as a product of degenerative changes in the nerve elements, peculiar fatty bodies appear in the perivascular spaces. These fatty bodies are pathognomic of hydrophobia: in no other disease have objects resembling them in size, form, and number hitherto been observed. They are identical with the "highly refractile hyaloid substance" of earlier authors, and are best seen in fresh preparations. (2) The inflammatory process is an early stage of acute myelitis or encephalitis. That it does not, like acute myelitis generally, go on to softening, depends on the short duration of the disease, this again being probably due to the localisation of the lesion. (3) The pathological changes were most marked in the medulla oblongata, especially in the nuclei of the eighth nerve. In the spinal cord, the parts most affected were the surroundings of the central canal, the anterior cornua, and the margins of the posterior cornua. (4) In the human brain, the process was confined to the spinal cord and medulla; in the dogs' brains, it was seen also in the corpora quadrigemina, the basal ganglia, and the cerebral hemispheres; it was frequently found in the olfactory convolutions, but very rarely in any other part of the hemispheres, and in the cerebellum it was never found.

On Combined (Primary) Disease of the Spinal Tracts.—Westphal contributes a lengthy paper on this subject to the *Archiv f. Psychiatrie* (viii. p. 469, ix. pp. 413 and 691). Five cases are reported: in four of them there was lesion of the posterior and lateral columns through the whole length of the spinal cord, the lesion in one instance extending to the anterior columns and the anterior cornua; in the fifth case (Schneider's) the lateral columns were implicated throughout their extent, but the posterior columns were unaffected in the lumbar region. These cases are considered respectively from an anatomical, pathological, and clinical standpoint.

The lesion of the lateral columns was in no case *systematic*; it was never confined to any of the systems of fibres into which Flechsig divides these columns. Whether the affection of the posterior columns was systematic is doubtful, and cannot be decided until we know more of the anatomy of this part of the cord. Fibres of different systems were, however, not unfrequently found affected in a portion of their course. Various attempts have been made to explain the localisation of the lesion. Some have

thought that the disease spreads directly from posterior to lateral column; but Westphal shows, by a careful examination of his own and other cases, that the lesions of the two columns are in a manner independent, and that the pathological process certainly does not extend directly from one to the other. Others have held that the initial lesion is a chronic inflammation of the posterior columns, which sets up a meningitis posterior; this again, by its extension, carrying the process to the lateral and anterior columns. A third view is that the lesion is simply a chronic myelitis, which, for some unknown reason, spreads chiefly in a longitudinal direction. This view, though beset with difficulties, is provisionally accepted by Westphal. Whether the lesion originates in any special focus or simultaneously affects the whole length of the strands is not known, but it would appear that the upper two-thirds of the dorsal part of the cord are specially concerned in its origin, for here the disease is often first observed and most severe. Westphal suggests that the peculiar distribution of the lesion may be due to the primary implication of the nerve-cells of the grey matter, for these are connected with the fibres of different systems. The author remarks that the lateral limiting layer of Flechsig (seitliche Grenzschicht der grauen Substanz) is always left intact.

Pathologically, the white matter showed two varieties of disease: (1) granular degeneration, or chronic myelitis; (2) grey degeneration, or sclerosis. The former, with one exception, was always found in the lateral columns, the latter always in the posterior columns. It must not be concluded, however, that the posterior columns never show the granular degeneration, for not unfrequently it occurs in quite typical cases of tabes dorsalis. But if the posterior columns exhibit granular degeneration, the lateral columns also invariably exhibit the same degeneration; grey degeneration of the lateral (or anterior) columns never co-exists with granular degeneration of the posterior columns. The relations of the two varieties have often been discussed under this form. Is the secondary degeneration of Türck (a granular degeneration) essentially the same as sclerosis (grey degeneration). Westphal holds that it is. He shows that the two are connected by intermediate stages, and sometimes are associated together; for instance, in typical cases of grey degeneration the sclerosed parts are not seldom surrounded by a small zone with all the characters of granular degeneration, from which it would appear that the granular is but the earlier stage of the grey degeneration.

Clinically, it is of interest to inquire in what way the symptoms

proper to lesion of the posterior columns are modified by lesion of the lateral columns. From his own four observations and from two others, Westphal concludes that where the lateral columns, and especially their pyramidal strands, are affected, the symptoms of ataxia are accompanied or replaced by those of paralytic weakness. This conclusion would seem to be stultified by a case reported by Friedreich, in which, though the lateral and anterior as well as the posterior columns were diseased, the muscular power remained intact. This case certainly shows that a considerable part even of the pyramidal strands may be affected without any symptoms of motor weakness, but is not conclusive against the position just taken, for the degenerative changes were not sufficiently intense to make the case a crucial one; e. g. in the inferior parts of the cord they were not very marked, and they were less advanced in the lateral than in the posterior columns. Westphal next discusses the view that the motor weakness in these cases is due to lesion of the posterior columns themselves. Leyden has published a case in which there was ataxia and great motor weakness of the lower extremities, and yet the posterior columns alone were degenerated. Leyden thinks that in this and similar cases the motor weakness is a pseudo-paralysis, and is to be attributed not to any particular anatomical lesion, but to a want of innervation or force of will. He finds support for his view in this fact, among others, that ataxic women present the symptom much oftener than men. Westphal does not accept this view. He points out how inapplicable it is to cases of one-sided paralytic weakness. This solitary case of Leyden does not, in his opinion, preclude us from holding that in the numerous cases of ataxia with motor weakness and lesion of the lateral or anterior columns, the lesion and symptom are connected as cause and effect. Lastly, Westphal discusses the theory that the motor weakness is caused by changes in the muscles themselves. His conclusion is that neither simple atrophy nor atrophy with degeneration satisfactorily explains the impaired motility. The author sums up this part of his paper by stating that the motor weakness in these cases is most probably produced by the lesion of the lateral or anterior columns, though, as yet, the more precise relation of the symptoms to the localisation of the lesion remains unknown.

Westphal's cases lend no support to Charcot's statement, that where there is lesion of the lateral columns, the paresis or paralysis is always accompanied by contracture. They show, on the contrary, that in combined disease of the posterior and lateral columns

muscular rigidity and spastic contracture are not observed, if the lesion of the posterior columns extends to the lumbar portion of the cord and affects the parts known as the root-zones. In three of the cases symptoms of affection of the trigeminus were observed, viz. paræsthesia, painful sensations in skin, pain in chewing, analgesia, lachrymation, and (in one case only) neuroparalytic keratitis. These disturbances of sensibility were associated with certain peculiarities of physiognomy, which are to be regarded as evidence of an ataxia of the facial muscles. The trigeminus presented the same pathological changes as the posterior nerve-roots; the atrophy of the ascending root could be traced into the medulla oblongata. Fits of reddening of the face, salivation, perspiration, dyspnœa, and enormously heightened rapidity of pulse were several times noticed, these symptoms being doubtless due to some bulbar lesion. In one case, where the sensibility of the skin was almost normal, and that of the muscles very considerably affected, the lesion of the posterior columns was localised in a peculiar manner; a strip of white matter bordering the posterior septum, the posterior commissure, and the posterior cornu was left intact. This case is interesting in connection with the supposed different courses taken by the sensory fibres of skin and muscle.

The diagnosis of combined disease of the posterior and lateral (or anterior) columns rests mainly on two symptoms; (1) the peculiar gait, which differs from that of an ordinary ataxic; and (2) the appearance of muscular weakness, with or without evident ataxia. But these symptoms are not pathognomonic; they may be present when the posterior columns alone are diseased.

Schneider's case presents several points of interest. There was pseudo-secondary degeneration of the posterior columns, and of the pyramidal and cerebellar tracts of the lateral columns; that is to say, though the lesion was a primary and independent one, it resembled a secondary degeneration in its distribution. At the posterior part of the cord there was a chronic leptomeningitis, to which perhaps the disease of the subjacent white matter owed its origin. Diabetes complicated the later history of the case; it was brought on apparently by faradisation of the limbs, but whether due to this or to disease of the cord is uncertain. Spastic spinal paralysis was observed. It differed from the typical form described by Erb in this, that there were disturbances of sensibility and slight affection of the bladder and sphincter ani, but it does not on this account cease to be spastic paralysis. Westphal concludes his paper by some remarks on this affection, pointing out that in

cases of combined primary disease of the spinal cord it may be associated with (1) pseudo-secondary degeneration originating in the dorsal region of the cord; (2) lesion of the lateral and posterior columns, if the latter remain unaffected in their lumbar portion; and (3) disseminated sclerosis of the spinal cord or medulla (Charcot).

W. J. Dodds, M.D., D.Sc.

Head Measurements. Dr. J. S. Wight, of Brooklyn, U.S. (*Archives of Medicine*, vol. ii. No. 2, Oct. 1879, New York), in an elaborate paper, gives the results of observations made on the heads of 21 males and 21 females (uneducated), and 21 males and 21 females (educated). The subjects were all adult.

His paper tends to show that, in both educated and uneducated, the brain of the male has a comparatively greater volume in the *anterior* part of the cranial cavity than the brain of the female, whilst the female brain has a comparatively greater volume in the *superior* part of the cranial cavity than that of the male; and the education in both sexes tends to increase the volume of the anterior part of the brain. He demonstrates that the volume and form of the brain of the uneducated male somewhat nearly resemble the volume and form of the brain of the educated female; while in relation to education the female shows greater variation, in the development of the anterior part of the brain, than the male.

He considers that the female brain shows as great a capacity for development by education as the male brain, and under similar circumstances of mental work and heredity would be fully equal to it.

He argues from this that females ought to receive a higher education, which he thinks would tend to the amelioration of the race. He believes his conclusions will be supported by the facts of imperfect development operating as causes of diseased conditions.

In making his measurements Dr. Wight uses a somewhat indefinite spot on each mastoid process as a centre of departure; and from one mastoid centre-point to the other measures five transverse arches, which he calls inter-mastoid arches.

(1) An anterior arch above supra-orbital ridges.

(2) A frontal arch over frontal eminences.

(3) A middle arch nearly over coronal suture.

(4) A superior arch nearly over vertex, and about parallel with axis of foramen magnum.

(5) A posterior arch over the occipital protuberance.

Three diameters:—

(1) A base line from one mastoid point to the other.

(2) The greatest transverse diameter.

(3) The greatest antero-posterior diameter.

He also takes the greatest antero-posterior circumference (just above frontal sinuses and occipital protuberance).

He makes a great point of the masto-frontal angle, which is included between the planes of the anterior and superior arches, and the size of which, he says, gives an important indication of the development of the brain.

The greater the masto-frontal angle, cæteris paribus, the greater the volume of brain in the anterior part of cranial cavity.

This masto-frontal angle is apparently included between the fronto-mastoid line and the base-line of the skull, though formation is not clearly described in the paper.

CROCHLEY CLAPHAM.

General Paralysis amongst Negroes.—The American *Journal of Insanity* for October 1879, records in the "Proceedings of the Association of Medical Superintendents" some highly interesting remarks on this subject. Dr. Bryce, of Alabama, stated that notwithstanding a large experience amongst insane negroes, he had never met with a case of general paralysis amongst them. Dr. Callender, of Tennessee, had no recollection of having seen in the African any type of disease resembling general paralysis of the insane; and Dr. Nichols, of Washington, said that having begun in 1855 to have the supervision of twenty coloured patients—a number now increased to fifty—he had never in that experience seen a case of general paresis. It was stated that Drs. Compton, Longworthy, and Wallace, had also affirmed that they had never seen a case of paresis amongst the coloured people of the South. Dr. Black, quoting from the last report of the Central Asylum for the Coloured Insane in Virginia, stated that four cases had been admitted into that institution previous to last year. It is evident that general paralysis is characteristically rare amongst negroes.

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END OF VOL. II.

LONDON: PRINTED BY WM. CLOWES AND SONS, STAMFORD STREET AND CHARING CROSS.